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ANNUAL MEETING, OCTOBER 11-16, 1953, CHICAGO

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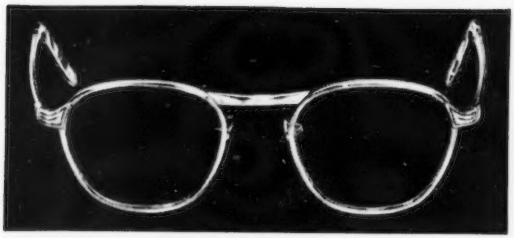

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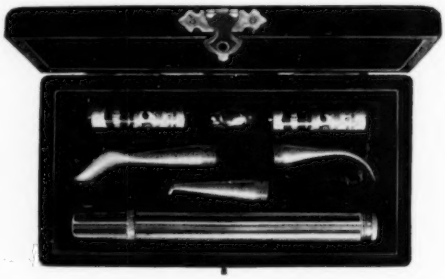

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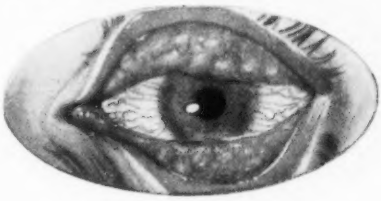

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2. Mitsui, Y., et al.: Antibiotics & Chemotherapy **1**:253 (July) 1951.
3. Dounas, N. G.; Featherstone, R. M., and Bradley, A. E.: A.M.A. Arch. Ophth. **46**:57 (July) 1951.

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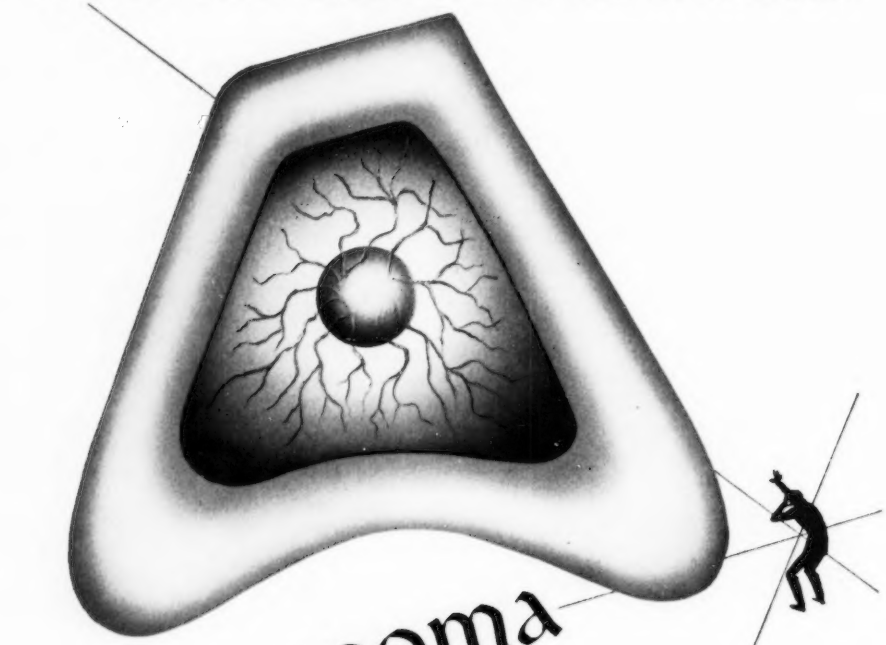

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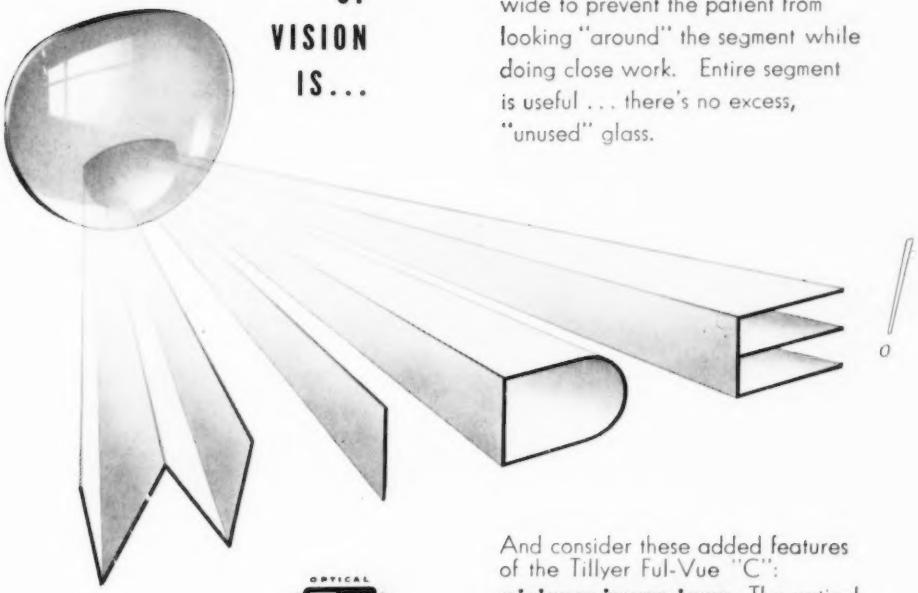

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1. Van Alyea, O. E., and Donnelly, W. A.: E.E.N.&T. Monthly, 31, Nov. 1952.
2. Fox, S. L.: AMA Arch. Otolaryn., 53, 607-609, 1951.
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6. Browd, Victor L.: Rehabilitation of Hearing, 1950.
7. Kugelmass, I. Newton: Handbook of the Common Acute Infectious Diseases, 1949.

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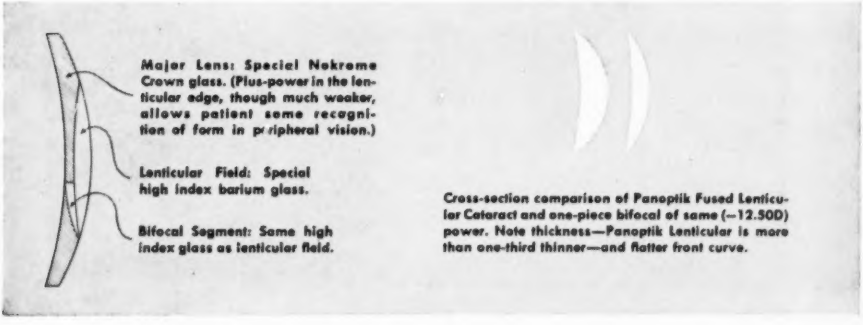
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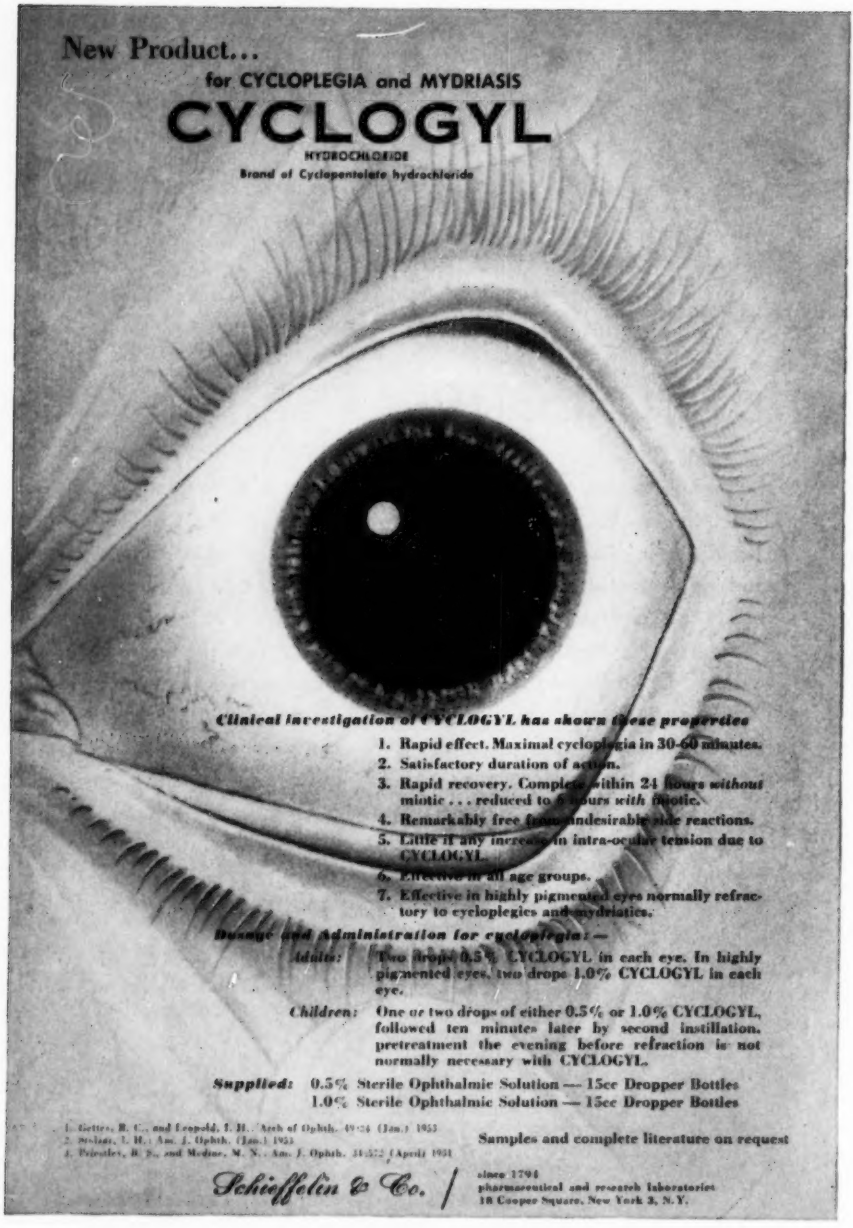
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1. Gettes, B. C., and Leopold, I. H.: Arch of Ophth. 49:24 (Jan.) 1953
2. Stulen, I. H.: Am. J. Ophth. (Jan.) 1953
3. Priestley, B. S., and Medine, M. N.: Am. J. Ophth. 35:572 (April) 1951
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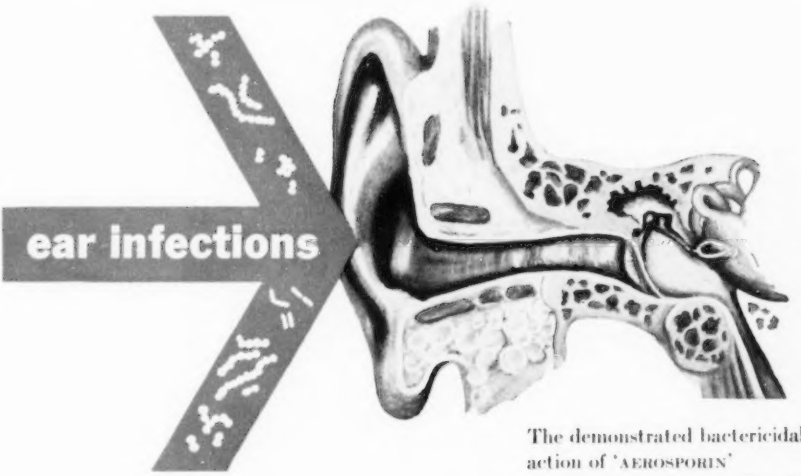

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2. Hayes, M.B. and Hall, C.F.: The Management of Otogenic Infection, Tr. Am. Acad. Ophth. 50:139, 1947.

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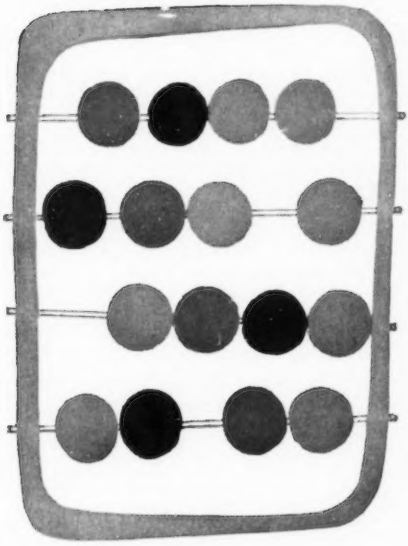

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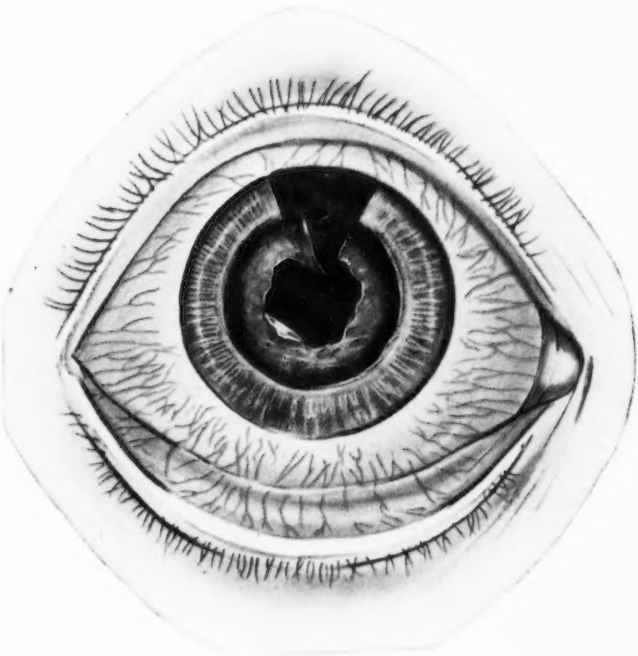

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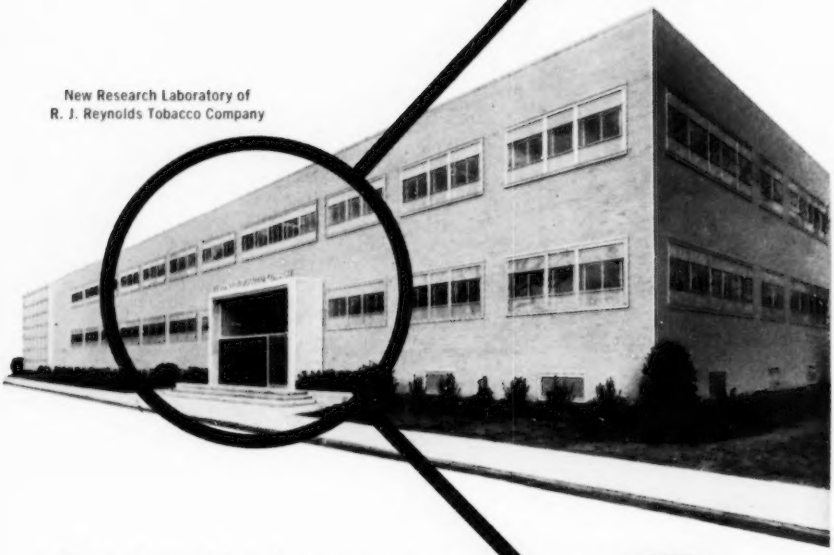

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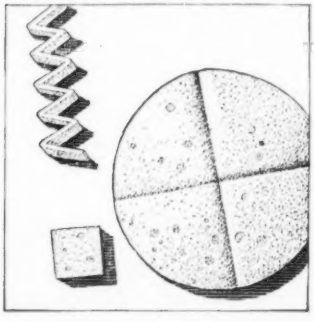

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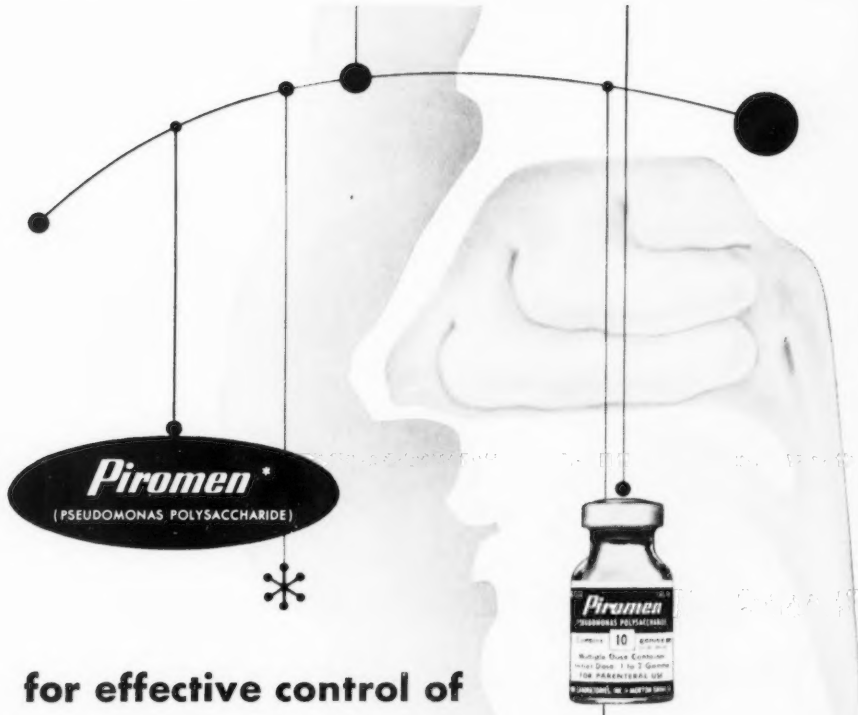

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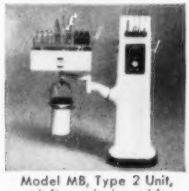

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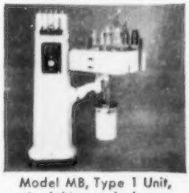

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TABLE OF CONTENTS

PAGE

VISUAL LOSS IN PRIMARY SKULL DEFORMITIES 497
BANKS ANDERSON, M.D., and
BARNES WOODHALL, M.D., by invitation, Durham, N. C.

OPHTHALMOPLEGIC MIGRAINE: A DISCUSSION OF ITS PATHOGENESIS WITH A REPORT OF THE PATHOLOGIC FINDINGS IN A CASE OF RECURRENT OCULOMOTOR PALSY 517
DAVID O. HARRINGTON, M.D., and
MILTON FLOCKS, M.D., by invitation, San Francisco, Calif.

FURTHER OBSERVATIONS ON THE SYNDROME OF GLAUCOMATOCYCLITIC CRISES. 531
ADOLPH POSNER, M.D., and
ABRAHAM SCHLOSSMAN, M.D., New York, N. Y.

PATHOLOGIC FINDINGS IN AN EYE WITH A NONHEALING CATARACT INCISION. 537
WILFRED E. FRY, M.D., Philadelphia, Pa.

SELECTED PROBLEMS IN THE DIAGNOSIS OF LARYNGEAL CARCINOMA 539
STANTON A. FRIEDBERG, M.D., and
LINDEN J. WALLNER, M.D., Chicago, Ill.

THE PATHOLOGY OF THE PHARYNGEAL RECESS, EUSTACHIAN TUBES, TONSILS AND ADENOIDS 555
HERMAN SEMENOV, M.D., Beverly Hills, Calif.

MEGAESOPHAGUS (ESOPHAGEAL DYSTONIA) AND ESOPHAGITIS 586
OWEN H. WANGENSTEEN, M.D., Minneapolis, Minn., by invitation

A NEW METHOD FOR OBTAINING EUSTACHIAN TUBE CLOSURE AT TYMPANOMASTOIDECTOMY 602
LOUIS E. ADIN, JR., M.D., and
DONALD A. CORGILL, M.D., McKinney, Texas

NEW INSTRUMENTS

ABSOLUTE THREE DIMENSIONAL COLORED RETINAL PHOTOGRAPHS 612
HERMAN J. NORTON, M.D., Rochester, N. Y.

INTRACAVITARY OPTIC RADON APPLICATOR 614
JOHN W. HENDERSON, M.D., Rochester, Minn.

PERIMETRY WITH ULTRAVIOLET (BLACK) LIGHT AND LUMINESCENT TEST OBJECTS: A PRELIMINARY REPORT 616
DAVID O. HARRINGTON, M.D., San Francisco, Calif.

"STERETAINER": A NEW DEVICE FOR DISPENSING STERILE OPHTHALMIC SOLUTIONS 620
RICHARD C. TROUTMAN, M.D., New York, N. Y.

TABLE OF CONTENTS *(Continued)*

PAGE

New Instruments 621
Oscar B. Nugent, M.D., Chicago, Ill.

New Capsulotomy Scissors 622
John M. McLean, M.D., New York, N. Y.

"Bed-Specs" 623
Clyde H. Jacobs, M.D., Danville, Pa.

Rhinophyma: Surgical Excision Following Oblique Cutaneous Incision 625
Gerson Lowenthal, M.D., Cincinnati, Ohio

Direct Vision Bone Cutting Forceps for Rhinoplasty 628
Irving B. Goldman, M.D., New York, N. Y.

A New Set of Adenoid Curettes 629

A New Laryngeal Dilator 631
Edley H. Jones, M.D., Vicksburg, Miss.

Tracheotomy Tube for Inhalation Therapy 632
Albert H. Andrews, Jr., M.D., Chicago, Ill.

EDITORIALS

Supplementary Report of the Board of Trustees Concerning the Resolution on Definition of Medical and Dental Service 633

Letter to the Editor 633

In Memoriam 634

FIFTY-EIGHTH ANNUAL MEETING

Official Notices 635
Convention Activities 642
Candidates 644
Program in Brief 653
Scientific Sections 655
Special Scientific Programs 671
Scientific Exhibits 674
Technical Exhibitors 679
Technical Exhibits XXIX
Section on Instruction XL
News Notes LII
Positions Available LIII
Available Supplies LIV

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VISUAL LOSS IN PRIMARY SKULL DEFORMITIES

BANKS ANDERSON, M.D.
and
BARNES WOODHALL, M.D.
BY INVITATION
DURHAM, N. C.

IT IS generally recognized that certain hereditary or congenital skull deformities may be associated with visual disturbance. That modern surgery offers hope of prevention of visual loss in such deformities is less widely known. This paper discusses the problem of the recognition of those primary deformities which in the early stages of maldevelopment may cause or be associated with chronic papilledema and in the later stages with optic atrophy, and describes a means of preventing this complication. Since, obviously, late recognition of chronic papilledema or atrophy implies organic change within the optic nerve, the first and perhaps the most important step in the prevention of blindness in these individuals is the recognition of or, in certain families in which the hereditary character of the disease is well established, the anticipation of the skull deformity. The eye of the alert and trained physician may catch the imprint of the skull deformity at birth or recognize the condition within the first year of life, whereas his less discerning colleague may not become aware of the abnormality until it is far advanced in development and may even then fail to appreciate the associated visual hazard. Early recognition of a skull deformity implies a certain degree of familiarity with the subject and an awareness of the implications of its appearance. In developing a background of general information which may serve to keep this condition in mind, one may profitably review the contributions of investigators with somewhat collateral interests.

Skull deformities are within themselves a fascinating field of study. Evidence of widespread and continuing interest in the subject may be found in the extensive bibliographic lists appended to the comprehensive papers of Enslin,[9] Greig,[16,17] Park and Powers,[29] Gunther,[18] and Ferriman,[13] as well as in the numerous more recent case reports. In developing this paper free use has been made of these monographs, in most cases without further reference to source material. Rather than encumber this manuscript with repetitious bibliography, it is suggested that those interested in documentation of any of the general statements which may follow refer to these exhaustive lists.

HISTORICAL BACKGROUND

In our more remote ancestry, skull deformities must have been the origin and focal point of many superstitions. Some mystico-religious concept founded on the spontaneously occurring deformity resulted in the widespread custom of producing artificial deformities in the infant by various pressure

From the Divisions of Ophthalmology and Neurosurgery, Duke University Medical School and Hospital.

Presented at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

mechanisms. This practice in one form or another appeared among almost all primitive peoples. Such artificially deformed skulls have been found many times in the ancient cemeteries of Hungary as well as in the graves of the pre-Columbian inhabitants of North and South America. In North America the custom persisted in certain Indian tribes of the Northwest until the early nineteenth century. The acceptance, prevalence and persistence of this practice in different peoples in all parts of the world is ample proof that increase in the vertical index of the skull within itself neither produces blindness nor dulls the intellect. On the contrary the high-vaulted head has always been considered a mark of especial distinction. The ancient Chinese god of prosperity, Fukurokuju, through prolonged meditation developed a fantastically high dome. Shou-Lao, god of long life, was similarly endowed. Let anyone doubting that men of genius are endowed with abnormally expanded crania recall that the most observant Sherlock Holmes described his Nemesis, Professor Moriarity, as a man whose "forehead domes out in a white curve and his eyes are deeply sunken in his head." Professor Moriarity, on the other hand, found Holmes to have "less frontal development than I should have expected." References to the spontaneously occurring deformity, according to Gunther, can be found in the Iliad, where Thersites was described as having a pointed head (Spitzkopf). A similar expression is said to be found in the Epidemics of Hippocrates as well as in the writings of Aristophanes. Ferriman, while doubting the interpretation of the Homeric reference, agrees that "acrocephaly is an ancient disease. The description given by Oribasius (A.D. 325-403) is astonishingly full." It is hoped that this sketch of the significance of skull deformities in historical and literary perspective may stimulate some latent interest or at least fix the picture in the reserves of one's mental files. With the resources of a memory so conditioned one may perhaps be more alert in the recognition of a deformity when encountered.

TERMINOLOGY — PATHOGENESIS — CLASSIFICATION

Present knowledge of this complex subject as well as a lamentable confusion in terminology derives from the chronologically overlapping investigations of anthropologists, pathologists, ophthalmologists, radiologists and geneticists, whose diversified interests have peaked at different intervals. The introduction of the relatively modern science of craniometry, with the introduction of new terms as well as the attempt to compress and adapt those already in common usage into a logical arrangement, has failed to clarify the confusion. The terms "dolichocephalic," "brachycephalic" and "hypsicephalic" signify to the anatomist and anthropologist development, in many instances as a racial characteristic, of the skull in length, breadth or height, without implying in any way the pathologic connotations which such terms suggest to the pathologist or surgeon. Virchow, in 1852, first used the expression "oxycephaly" to describe a type of deformity characterized by extreme upward growth with reduction in lateral and anteroposterior diameters. Lucea, according to Gunther, in 1847 first used the term "acrocephaly," an expression of indefinite antiquity and loose usage, as synonymous with the tower skull or oxycephaly of Virchow. Unfortunately, at about this same period anthropologists applied the term "acrocephalic" to a large class of normal skulls of certain index. Various other descriptive terms have since ap-

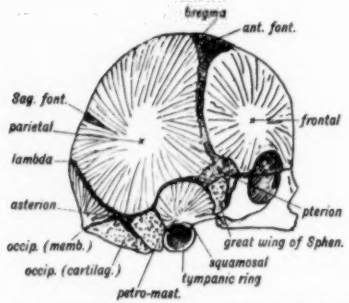
peared, such as turricephaly, scaphocephaly, trigonocephaly, plagiocephaly. All refer to a particular pattern of deformity expressed principally in the calvarium.

In 1924 Greig introduced the term "hypertelorism" to describe a deformity characterized by excessive interocular width. This type of deformity had previously been reported, according to Berliner and Gartner,[5] by Fidolin as a variety of plagiocephaly. Greig, on the basis of two autopsy studies, realized that the wide interorbital space was due to a different and hitherto undescribed mechanism which justified the claim of a new syndrome. More recently another expression, "platybasia," has been introduced to identify abnormalities in the occipital bone. The problem of a meaningful terminology and rational etiologic classification becomes more difficult, the result more confusing, when an attempt is made to integrate the deformities of the vault and base of the skull with the allied deformities of the face—the craniofacial, the cleidocranial, the mandibulofacial dysostoses—as well as with the associated deformities of other mesodermal tissues, the syndactylias and synarthroses. Furthermore, from the diagnostic point of view the acquired deformities and fringe syndromes, the lipochondrodystrophy of Hurler, infantile cortical hyperostosis, rickets and probably many other less well-known syndromes and diseases must receive consideration.

Rather than attempt to bring order into such an extensive list in which the nomenclature reflects external configuration, a better understanding of interrelationships may be attained by establishing a possible common denominator over which this wide range of deformities can be integrated on a causal basis. Sir Arthur Keith,[22] celebrated curator and anthropologist of the British Museum, has shown how many obscure developmental processes in the human can be best understood in the clarifying light of comparative anatomy. The generalization that ontogeny recapitulates phylogeny leads to an understanding of the development of the normal skull as well as its abnormal variations. The amphioxus brain, if it may be designated as such, is encased in an exclusively mesodermal membranous sheath. In selachians (dog fish) the brain has enlarged and cartilaginous plates have developed in its expanded investment, forming a chondrocranium. In teleosts (bony fish), higher in the evolutionary order, dermal bony plates appear on the sides and vault of this primitive but greatly expanded cartilaginous capsule. In the orderly processes of evolution the mammalian skull is similarly derived from the mesodermal membranous investment of the brain. In the ventral aspect of this membrane, as in the dog fish, cartilaginous plates appear and fuse into the chondrocartilaginous base of the skull. However, the process of chondrification extending from the base along the sides to the vault does not keep pace with the demands of the rapidly growing cerebral vesicles.

The dermal plates which first appeared in the teleost in association with cartilage find no such supporting structure when they appear in the expanded mesodermal sheath surrounding the embryonic mammalian brain. These plates therefore develop independently and become the centers of ossification of the membranous bones of the skull. The walls of the skull are thus made up of two separately derived elements, basilar and capsular. The growth of the base of the skull, the chondrified portion, is determined not only by the demands of an expanding brain (the neurocranial element) but also by the needs of the facial, olfactory, and masticatory components (the splanchnocranial ele-

ment). In the basal portion growth proceeds as in the long bones, the lines between the basioccipital and basisphenoid, the basi and presphenoid and between the presphenoid correspond to the epiphyseal lines of long bones. The base of the cartilaginous skull is encased in a perichondrium continuous with the



The Centres of Ossification for the Dermal Bones of the Skull. The bones which are formed in cartilage are stippled.

FIG. 1.—From Keith, Arthur: Human Embryology and Morphology, ed. 6, London, Edward Arnold & Co., 1948.

membranous capsule. In this membranous capsule, as has been explained, on each side appear four centers of ossification for the four cranial bones developing in this structure (fig. 1). As the osteoblastic fibers and bony spicules develop and extend from these centers, the surrounding membranous capsule is split and separated, producing the periosteal layer within and the pericranial layer without. However, beyond the advancing bone the capsule remains fused, forming a mesenchymal sutural gap or septum which provides the matrix into which grow the fibroblasts and osteoblasts from which bone is derived.

Growth in the coronal and lambdoidal sutures adds to calvarial length. Growth at the sagittal and squamous sutures increases the breadth. In the deformed skull there is a failure or premature absorption of this sutural septum permitting direct contact of the edges of adjoining bones. Where such bones touch, fusion occurs. The growing brain demands compensatory expansion of the skull which, being incapable of extension along one sutural line, responds by greater growth at another unsynostosed line. It is this underlying defect in the suture gap which predicates a deformity — the particular pattern of the deformity depends upon the particular suture line involved in the synostotic process.

There is evidence that in addition to the abnormal interosseous septum which makes possible early fusion, there is a primary aplasia of the bones themselves. The primarily affected bones may be flattened and the frontal and parietal eminences may be lacking. The bones themselves, according to Ferriman, are dense and smooth and the diploe are absent — digital markings or convolutional atrophy are a prominent feature. Since the skull of an infant in which the bones are not aplastic, as in infantile idiopathic hydrocephalus, may be enormously expanded without these markings, their presence in synostoses is considered additional evidence of aplasia. This primary aplasia is probably responsible for the underdevelopment of the splanchnocranium, the external evidence of which may be manifest in the orbit, maxilla and mandible. However, this aplasia plays little or no part in the causation of the increased intracranial pressure which is responsible for injury to the optic nerve. Evidence continues to accumulate that the underlying defect is primarily in the germinal mesoderm and probably is, in the great majority of cases, an inherited characteristic, a phase of the problem reserved for later consideration.

If one accepts the fact that the underlying defect in the developmental anomalies is in the germinal mesoderm

and hereditarily conditioned, a common denominator emerges which brings into focus the various diversified morphologic patterns. When Virchow first explained the various skull deformities by premature fusion of sutures, he pointed out that when a cranial suture is fused, growth must take place coaxial to the line of fusion. Application of this rule explains the various patterns of deformity. The severity of the optic atrophy as well as the degree of brain injury varies directly in proportion to the time and the completeness of the premature fusion of the membranous bones in relation to age and brain growth and inversely with the magnitude of skull expansion of whatever pattern permitted by the extent of the synostosis. When the coronal suture is prematurely fused, growth takes place in the intraparietal and occasionally in the interfrontal lines and is therefore directed mainly upward (oxycephaly). Exaggerated upward growth occurs when the frontoparietal and occipitoparietal sutures fuse synchronously (turricephaly). In scaphocephaly the premature fusion occurs in the sagittal suture—growth upward is impossible—overdevelopment in the long axis produces the dolichocephaly. Trigonocephaly describes excessive development of the posterior portion of the skull with maldevelopment of the anterior portion.

Closely allied with these deformities primarily expressed in the calvarium is a group which exhibits in addition to the calvareal deformity certain facial characteristics associated with aplasia of the facial bones—the craniofacial dysostosis of Crouzon.[7] Also a group specific syndrome has been built up (Apert's syndrome[1,2]) consisting of syndactylia in addition to the acrocephaly. Hypertelorism was a term introduced by Greig[16] in 1924 to describe the widened interocular space associated with the greatly enlarged lesser wings of the sphenoid and presumably due to a defect in the mesodermal anlage of the chondrocranium. Optic atrophy has been rarely seen in this condition and is probably due to traction or torsion of the nerve rather than to papilledema.

All of the three latter deformities, the craniofacial dysostosis of Crouzon, the acrocephalosyndactylia of Apert, and the hypertelorism of Greig, have had the finer facial characteristics submerged in gross physical changes which are so uniform in each syndrome that each group appears species specific. Platycephaly refers to an enlarged and overhanging posterior fossa impinged, as it were, on the cervical vertebra. Owing to the basilar invagination, the odontoid process of the epistropheus may enter the narrowed foramen magnum and come into close relation with the medulla oblongata (platybasia). Platybasia is a true primary deformity, probably conditioned by the aplasia or premature fusion of the membranous components of the occipital bone. The symptoms are mostly referable to cord pressure, but eye signs, particularly nystagmus and optic atrophy, have been observed. Decompression operations have been devised, the character and technic of which are considered beyond the scope of this paper. Hypertelorism and platybasia have been admitted to this extent in the discussion in order to present the full picture of the major primary deformities. In this paper no further consideration will be given these two conditions.

FREQUENCY OF PRIMARY SKULL DEFORMITIES AND IMPORTANCE AS A CAUSE OF OPTIC ATROPHY

We have been unable to find any study of the frequency in the general population of the deformities under consideration. In the 1940 report of the

causes of blindness among recipients of the aid to the blind, Hurlin, Saffian and Rice[19] estimated the total blind population of the United States at approximately 230,000. Some 30,000 of these individuals were judged to be blind as a result of optic atrophy. In an analysis of the causes of optic atrophy in children Cordes[6] found approximately 5 per cent (4 in 81 cases) attributable to cranial synostosis. In the North Carolina School for the Blind we found two acrocephalic skulls in 25 cases of optic atrophy. In approximately 50 per cent of Cordes' cases the atrophy was attributable to some type of hereditary or congenital disease. It is interesting that this proportion parallels the generally accepted idea that 50 per cent of all blindness is hereditarily or congenitally conditioned. If we accept the premise that one half of the 30,000 individuals blind because of optic atrophy represent hereditary or congenital disease and interpolate the 10 per cent value derived from Cordes' 4 cases with synostosis in 41 cases of optic atrophy hereditarily or congenitally conditioned, we arrive at the interesting but, we hasten to add, not necessarily accurate figure of 1,500 individuals blind from synostosis in 1940, an incidence of 1 to 70,000 in the general population. As is shown in this paper, approximately one third of the synostoses, all types considered, are complicated by an optic atrophy. We may therefore estimate that 1 in 20,000 of the general population is affected with a synostotic deformity. This incidence is somewhat less than one would estimate as a rough guess based on one's recollection of the relative frequency in which the condition might be encountered in daily social and professional contacts. In the Duke Hospital series there were 6 cases from the immediate environs of Durham, a community with a 100,000 population. These 6 cases certainly do not represent more than a third of the total number of cases, so the incidence is probably nearer 1 in 10,000. The occurrence of such an anomaly so infrequently explains and excuses the failure of the general practitioner to identify the disease. As Greig has stated, "Oxycephaly is so uncommon that no observer can record from personal experience many cases of it." However, in special fields of medical practice, particularly in pediatrics, ophthalmology, radiology and neurosurgery, the condition is much more commonly encountered. In the Roentgen Institute of Vienna, Dr. Arthur Schuler noted turricephaly 67 times and scaphocephaly plagiocephaly 10 times in 5,000 skull examinations.[18,28] Since skull roentgenograms would hardly be ordered unless some abnormality of the head were known or suspected this ratio of 15 synostoses in 1,000 routine skull roentgenograms is obviously much too high for the general population but does approximate the frequency with which the condition may be encountered in hospital practice.

As Virchow (1852) is credited with first having recognized the skull deformity as a result of premature synostosis, so should Von Graefe (1866) be credited with having first realized that this premature synostosis was responsible for visual loss through an associated papilledema and optic atrophy.

Optic atrophy is infrequently encountered in scaphocephaly. This is to be anticipated since premature synostosis of the coronal and lambdoidal sutures seems to curtail expansion more critically. Involvement of this middle area of the skull with lateral and upward expansion is most frequently associated with papilledema and atrophy. In 30 cases of Apert's syndrome (acrocephalosyndactylia) collected by Park and Powers, papilledema or atrophy was

present in 7. The discs were normal in 2 and not described in the remainder. Ferriman did not differentiate between Crouzon's craniofacial synostosis and acrocephaly in his report of the involvement of the optic nerve 146 times in 304 cases. Beyond doubt, many cases escape notice until they come to the ophthalmologist for loss of vision, and accurate figures are therefore impossible to obtain. However, Fletcher,[14] in his careful review, says of the vision in oxycephaly: "This is occasionally unimpaired, but in the vast majority of cases sight is very defective." Sight is, as a rule, impaired quite early in life. Meltzer's tabulation of 20 cases illustrated this point[10]:

	DEFORMITY FIRST NOTED	BLINDNESS FIRST NOTED
At birth	13	1
During first year	..	1
During second year	7	1
During third year	..	10
During fourth year	..	5
During fifth year	..	1
During sixth year	..	5

Of the 146 cases of optic nerve involvement in the 3 or 4 records collected by Ferriman, papilledema was observed 15 times; of the remaining cases in which the fundus could be visualized, 82 had either postpapilledemic atrophy or actual papilledema, and 22 had the type of atrophy found with direct pressure. According to Ferriman, papilledema and postpapilledemic atrophy clearly point to raised intracranial pressure but do not exclude some form of direct pressure from being present as an additional factor. He is of the opinion that raised intracranial pressure, narrowed foramina, and some form of localized pressure, such as kinking of the nerve or pressure from the carotid artery, all are definite causes of optic atrophy. In some cases two or more causes have been operating at the same time. Our evidence is against this generally accepted view. The neurosurgical group in general holds that the only factor involved in the optic nerve injury is the increased intracranial pressure. Optic atrophy has been reported in all of the various groups of the synostoses but appears to be much more common and complete in the group in which expansion is vertical, i.e., oxycephaly, turricephaly, tower skull.

HEREDITY AS AN ETIOLOGIC FACTOR

Most English, American and French students of genetics are of the opinion that the various primary dysostoses and synostoses are hereditary diseases. Ferriman does not concede that Crouzon's dysostosis craniofacialis is distinct from acrocephaly. He has collected 44 cases of acrocephaly in which the inheritance is claimed. On the basis of the evidence offered in these reports, he states that there can be little doubt of these claims in 22 families. As part of the evidence presented, he cites the appearance in four sets of monozygotic twins, those of (1) Engerth, (2) Gundrom, (3) Laignel, Lavestine and Boquier, and (4) Oeller. The significance of these twin studies in establishing the hereditary character of the disease will be discussed later. Ferriman reports the involvement of 2 generations of acrocephaly in 9 families. Claims are made for the involvement of 3 generations in 5 families, and of 4 generations in 1, although the published evidence is conclusive in none beyond the two-generation level. Unquestionable familial transmission of acrocephalosyndactylia has been reported four times. Evidence of inheritance is presented in another 10 families. Ferriman quotes Dr. L. S. Penrose as interpreting his data on the hereditary characteristics of this disease.

Both acrocephaly and acrocephalosyndactylia seem to be examples of dominant heredity. The clinical appearance of a rare recessive disease in two consecutive generations is extremely uncommon and almost unknown in three. . . . The relation of acrocephalosyndactylia to acrocephaly is obscure but the most logical explanation here would appear to be that two allellomorphic genes may be at work, one leading to acrocephaly and the other to the more severe acrocephalosyndactylia, modifying factors again being at work leading among other things to suppression of joint changes in some of the cases of acrocephalosyndactylia.

Of interest in supporting such an interpretation is the report of acrocephalosyndactylia with cleft hand in uniovular twins by Goll and Kalich-Koenner.[15]

Sorsby[30] implies acceptance of the hereditary nature of oxycephaly when he states, "The affection is said to be commoner in males. Most cases are sporadic but there are records in twins, in sibships, and over two and even three generations." Of acrocephalosyndactylia, he says genetically some pedigrees suggest recessive inheritance and others dominance, perhaps irregular dominance. Of craniofacial dysostosis, "Inheritance is more clearly dominant than in the Apert syndrome." Of hypertelorism, "Dominant inheritance has been frequently recorded." Larsen[27] found the deformity present in 16 males and 3 females in the cases he studied. He calls attention to a similar sex distribution in the cases of Patry, 58 men and 10 women, and offers this distribution as evidence of the hereditary character of the disease.

Greig[16] takes no positive stand on this question of heredity. He does state that there are no proven cases in 3 generations but incorporates in his paper the case reports of Butterworth in which 2 brothers and a grandfather were said to be affected, the report of Stevenson in which the mother and 2 of 5 children were affected, and of Chatlin in which the mother and child were affected. He also mentions in the same paragraph the case of a Dr. John Eason in which a boy with delayed oxycephaly had a normal twin brother. In this connection it seems worth while to explain that Greig, in speaking of oxycephaly, describes three types of the disease: (1) the true, recognizable at birth and due to general craniofacial synostosis associated with syndactylia and other deformities; (2) delayed, manifest in childhood and not associated with deformities of the extremities; and (3) false or localized cranial synostosis occurring during the cerebral growth, not congenital and often of definite origin. While taking no positive stand, Greig tends to stress more the congenital as contrasted with the inherited character of the disease.

Gunther[18] takes a diametrically opposite position from that expressed by Ferriman.[13] His opinion, backed by extensive investigation, demands respect. Although recognizing the occurrence of the disease in siblings, he discounts, at least to his own satisfaction, this occurrence as evidence of a hereditary factor. He makes much of the occurrence of sporadic cases in which "the consanguinity of parents plays no significant role." He is very positive in the statement that "the tower skull is no genetic constitutional anomaly." He discounts the predominance of the deformity in the male by claiming that it is so much less easily recognized in the female by reason of the abundance and arrangement of the hair. Whereas other investigators have offered the occurrence of the disease in monozygotic twins as evidence of a hereditary factor, Gunther submits the reports of Vonschuer and Siemans in which the deformity occurred in only one of identical twins. The following twinships in which both were affected were reviewed: Oeller, 1 case; Vonschuer, 2 cases; Marque, 1; Hanhart, 2 identical of triplets affected, the third was nor-

FIG. 2.—Monozygotic acrocephalic (oxycephalic) twins.

mal. Also reviewed by Gunther were twinships in which only one of the pair was affected: Eskuchen, Mirananoff, V. Vonschuer, Siemans. Unfortunately it is not clear whether these are monozygotic or dizygotic. After analysis of a series of twinships reported by Vonschuer and Siemans, in which were included all reported cases of acrocephaly, Gunther states that in both monozygotics and dizygotics one twin developed the deformity in approximately 5 per cent of the total number of cases reported. Gunther makes much of the fact that, as reported by V. Vonschuer, when only one twin is born with a tower skull, this is always the second born.

This rule was encountered in 7 cases of the observances of Vonschuer and supports gynecological experience that in the monozygotes the lighter and thereby the growth-damaged child is always the second born. The twin studies permit the deduction that rather than hereditary factors, one must consider the possibility of intra-uterine injury to the germ plasm. All clinical experience supports the assumption that only in an insignificant part of the tower skull cases abnormal hereditary factors could have a genetic importance.

This question of etiology is of some importance. If we are dealing with a hereditary disease, the accumulation of sufficient data over a period of years may enable one to plot, as it were, the possibilities of its appearance in certain families. Optic atrophy may be prevented by anticipation. Also if the hereditary nature of the deformity can be definitely established, one can authoritatively negate the shadowy suspicions and self-incriminations of these unfortunate mothers, at least in so far as any feeling of personal guilt is concerned.

Supporting the concept of a hereditary etiology we wish to present two families whom we think demonstrate the genetic transmission of the deformities. These cases also show by contrast what can be done towards preserving vision.

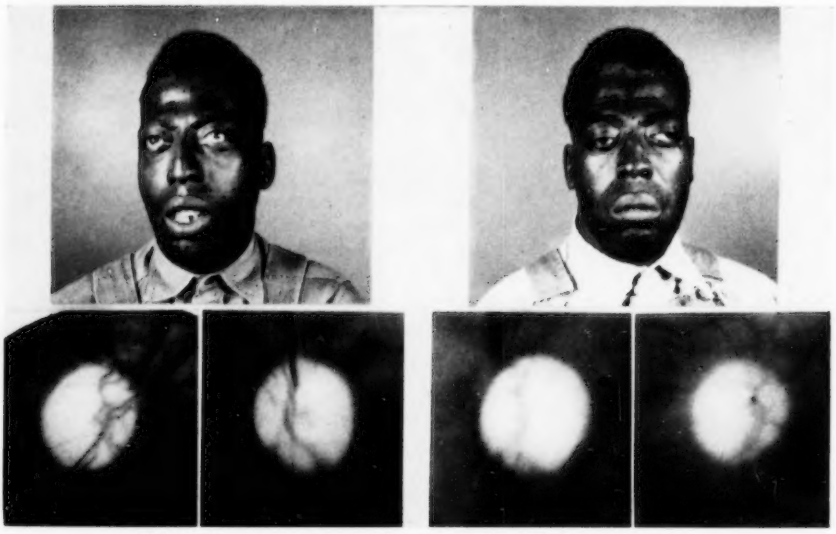


FIG. 3—Monozygotic acrocephalic (oxycephalic) twins with optic atrophy.

FIRST FAMILY

These 33-year-old monozygotic twins (fig. 2) were first seen in 1932. One of the twins was brought in unconscious from a skull fracture resulting from a fall. The past history of this patient was entirely noncontributory except for loss of vision extending back beyond memory. The other twin had no significant past history other than visual loss also extending beyond memory. Vision in both cases was limited to light perception. The details of the pregnancy were not available. The boys were presumably born at term; development was normal. The children were walking in the first 2 years, talking at 18 months. Physical examination of both twins was completed after recovery on the part of one from the above noted skull fracture. The appearance of the skulls was very striking. One was immediately impressed by the complete identity of all minor characteristics such as shape and size of ears, and the lines characterizing the face. The fingers and palm prints were also in the same general grouping (complete similarity of finger prints does not occur in monozygotic twins). The skulls were constricted both anteroposteriorly and laterally, extending upward and backward with a sharply slanting forehead. At the peak the configuration was somewhat truncated and to an equal degree in both skulls representing an extreme form of oxycephaly. The eyes of both were markedly exophthalmic, the lids thickened and heavy. Pupils of both were regular and reacted to light. There was external deviation of the eyes in each individual of no particular pattern. The optic papillae in both showed marked atrophy (fig. 3). The hard palate and nose showed no particular deformity. Physical examination was otherwise normal. Roentgenograms showed complete closure of all the suture lines and almost complete obliteration of the anterior fossa. There was marked convolutional atrophy throughout (fig. 2).

The family history was negative for similar or like abnormality; two siblings showed no evidence of deformity.

In June 1952 the condition of each remained essentially the same as described in 1932, i.e., marked upward truncation of the skull, exophthalmos, external strabismus. Headaches, which had never been prominent, had disappeared completely; there had at no time been convulsions.

SECOND FAMILY

The second family in contrast to the twins described above demonstrates how vision can be saved in premature synostosis. The definite evidence of hereditary transmission with the characteristic involvement of the facial as well as the cranial bones places this family in that group described by Crouzon as hereditary craniofacial dysostosis. The cranial deformity is the oxycephalic type.

FIG. 4—Familial craniofacial dysostosis of Crouzon.

The first member of this family (fig. 4A) was referred by Dr. J. W. McLean of Greenville, S. C. The child, aged 5 on admission to the Duke Hospital, was born at term after an uncomplicated pregnancy and labor. No particular significance was attached to the prominence of the eyes, noted at birth, since this was considered a characteristic inherited from the father. As the child developed, the eyes gradually became more prominent. At 2 years of age one eye for a short time became spontaneously dislocated. The recurrence of this phenomenon two weeks before admission first prompted the parents to seek medical care. The child was considered to have developed normally otherwise, both physically and mentally. There had been some complaint of photophobia and epiphora, but no headaches of significance nor convulsions had been noted. On examination the skull deformity was obvious. The exophthalmos was conspicuous, as was the ocular divergence. The right eye was more proptosed than the left with a tendency to dislocate on the slightest pressure. The exophthalmos was estimated at 28 mm. on the right and 26 mm. on the left. The interpupillary distance was 70 mm. Ocular divergence was most conspicuous on the right. The lids were rather thick and somewhat leathery. The forehead was broad and rounded. The face was concave with underdevelopment of the maxilla. The nose was thin and slightly beaked, the upper lip shortened, the mandible overshot with moderate malocclusion. The palate was highly arched. The cornea measured 14.5 mm. in diameter. Both discs were edematous and elevated. The electroencephalogram recorded generalized dysrhythmia without evidence of localized disturbance. Roentgenograms of the skull showed marked convolutional atrophy and confirmed the underdevelopment of the maxilla (fig. 5A). The suture lines were all closed. The orbits were very shallow. The preoperative vision was 20/70 in the right eye and 20/100 in the left. Peripheral fields, while not reliable, indicated some contraction. Corrective surgery was undertaken in March 1948. In June 1952 vision was 20/20 in each eye. The discs though pale showed no elevation. There was some glial proliferation in the cups but the margins were clearly outlined. The peripheral fields were full. The exophthalmos was reduced to 24 mm. on each side.

The father, aged 27 (fig. 4B), was unaware of any difficulty other than an external strabismus and a reduced visual acuity. He was unaware of any visual disturbance or cosmetic blemish in his parents. His interpupillary distance was 78 mm. The corneas were 16 mm. in diameter. Vision was 20/40 in the right eye and 20/20 in the left. The eyes deviated outward about 15 degrees. The exophthalmometer reading was 25 in each eye. Both discs were slightly pale with blurred margins. The peripheral fields were normal. The

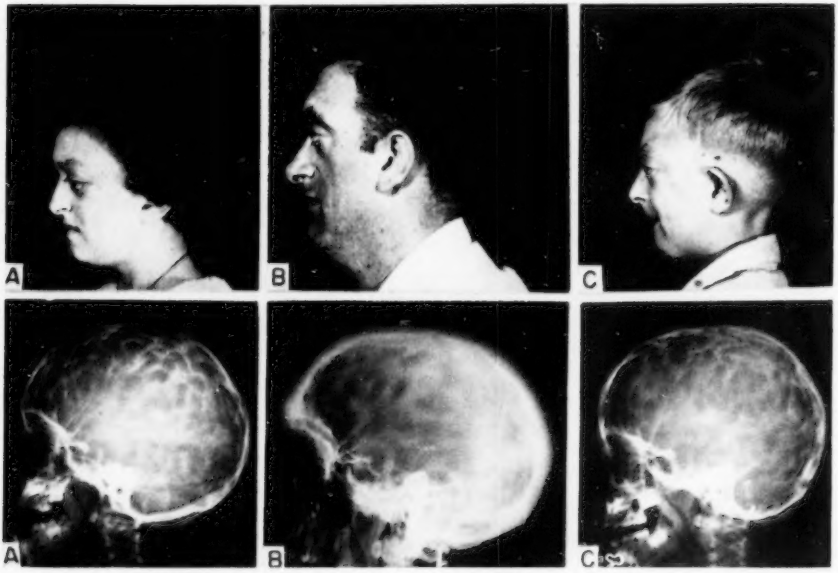


FIG. 5—Familial craniofacial dysostosis of Crouzon.

basis for the ocular difficulty was demonstrated by roentgenogram (fig. 5B). Suture lines were all obliterated. Increased convolutional atrophy or digital markings were conspicuous in the anterior portion of the skull. The frontal sinuses were absent. The orbital walls were angulated sharply downward. The orbits themselves were quite shallow. The palate was highly arched. The maxilla was underdeveloped, with protrusion of the lower jaw and moderate malocclusion.

The 4-year-old son of the patient just described (fig. 4C), by chance accompanied the father and sister to the clinic. He had no complaints. His development had been considered normal, although the parents suspected that he might be mentally retarded. His physical development had been normal. He was examined entirely because of the suspected familiar character of the disease. His skull was obviously deformed, with foreshortening of the anteroposterior diameter. The palate was arched, with underdevelopment of the maxilla, relative prognathism and malocclusion demonstrated by roentgenograms, which also revealed a convolutional atrophy of the skull with closure of all the cranial sutures (fig. 5C). The eyes were proptosed. Exophthalmometer readings could not be obtained prior to operation. The interpupillary distance was 70 mm. There was a divergent strabismus. There was moderate bilateral papilledema. Vision could not be accurately recorded but seemed not greatly impaired. Visual field studies were impossible. Operation for relief of increased intracranial pressure was performed in July 1949. In June 1952 corrected vision in the right eye was 20/30 and 20/20 in the left eye. The papilledema had subsided without significant secondary atrophy, irritability had disappeared and mentality had improved.

DIAGNOSIS

The signs and symptoms of premature synostosis like the pattern of skull deformity vary with the sutures involved and with the degree of involvement. Scaphocephaly is much commoner but rarely produces eye signs. Oxycephaly, as shown in this review, is accompanied in at least a third of the cases by involvement of the optic nerve. Papilledema may develop in the presence of an open fontanel. It has been observed by A. J. Ballantyne in a 4-month-old infant with synostosis. Exophthalmos is more prominent in oxy-

cephaly but is present to some degree in all types of synostosis. Exophthalmos is caused partially by the diminution of the depth of the orbit from the forward thrust of the middle fossa and partially by venous engorgement. The regression after surgical intervention is probably due to relief of venous embarrassment by lowering the intracranial pressure. A divergent squint is frequently present. The appearance of a knob in the region of the closed anterior fontanel is said to be an early and, when present, infallible sign. The beaked nose "parrot face" in Crouzon's craniofacial dysostosis, and the syndactylia with the marked brachycephaly, concave "dish" face and nose retroussé in Apert's acrocephalosyndactylia are, of course, within themselves diagnostic signs of great value.

The child with a synostotic skull is usually irritable, and has scholastic and social difficulties. Undoubtedly many cases classified as mentally defective fall in this group. Headaches and vomiting occur with great frequency, and convulsions are not uncommon. Convulsions are thought to be due to increased intracranial pressure in childhood. When occurring in middle life they may be associated with impairment of venous circulation. Superficial veins of the face and scalp, prominent before, have subsided after operation.

THE SURGICAL TREATMENT OF CRANIOSYNOSTOSIS

Although the underlying cause of this condition is inadequately understood, the primary pathologic physiology of suppression of brain growth by a mechanical skull disturbance is well recognized. The abnormal fusion of skull bones, occurring before birth or during the first few months of life, coincides with the onset of accelerated brain growth characteristic of the first years of life. The normal brain weight of 350 grams at birth is doubled at 9 months of age, tripled by the age of 2 years, and by the age of 5, four fifths of the adult brain weight of 1,400 grams has been attained. From a consideration of this growth curve, it is obvious that unrestricted brain growth is of particular importance during the first two years of life. The diagnosis of premature closure of suture lines should be established and surgical treatment initiated during this time period to prevent the development of the signs of mechanical compression of the brain that have already been described. Clinical experience has indicated, however, that much can be done to ameliorate the signs and symptoms of brain compression even in well-established instances of this condition in the older age groups. In general terms, all cases of craniosynostosis at any age level are fit candidates for surgical intervention.

The usual lack of any intrinsic pathology in the brain and the apparently simple problem of skull decompression which is involved stimulated efforts at surgical treatment. Indeed, Fairman and Horrax[12] have noted that palliative subtemporal decompressions were first performed by Krause in Berlin and by Eiselsberg in Vienna, and that such procedures were common in Cushing's clinic in 1913. Isolated attempts at curative linear craniectomy were reported by Lannelongue[26] in 1890 and by Lane[25] in 1892. The 2 patients chosen by Lane were imbecile infants; only one survived operation and the procedure fell into disrepute. Prior to 1924, with these exceptions, operations for craniosynostosis were done after irreparable changes had developed. Subtemporal decompression might relieve headache due to increased intracranial pressure and might prevent the progress of visual loss, but it was ill-suited technically for the problem of prophylactic control of

increased intracranial pressure in the young infant.

In 1924, Faber and Towne[10] advocated a revival of the so-called Lane-Lannelongue linear craniectomy and subsequently reported 5 cases in which the operation had been performed sufficiently early in the infant's life to prevent the usual sequelae of the condition.[11] Their first publication stimulated further efforts in this direction, and of chief interest is Bauer's circular craniectomy of the skull,[3] which was modified by Bennett, Keegan and Hunt[4] so that a distance of some 3 cm. was spared in the occipital region of the skull to ensure some fixation of the skull cap.

King, in 1938, presented a fresh approach to this problem by devising an operative technic in which the skull was made into a mosaic of square fragments, the operation being performed as a single or stage procedure.[23,24] The morcellation of the skull appeared to be a physiologic solution to the problem since it allowed brain growth to proceed in all directions and the results of this procedure were carefully studied by us.[31] Both King's cases and our own represented well-established instances of the condition and we did not have an opportunity to attempt this operation in the relatively soft plastic skull of the infant. The procedure was later modified by Fairman and Horrax[12] for the control of a single case of brachycephaly. This progress towards some type of linear craniectomy was interrupted briefly by Dandy's report of a hinged bone-flap to be used as a substitute for subtemporal decompression on one or both sides of the skull in a case of scaphocephaly.[8]

The early portion of a ten-year follow-up study of 5 cases of craniosynostosis in the older age group showed that even an extensive morcellation of the skull could not prevent rapid regrowth of bone and fixation of the pre-existing skull deformity. Indeed, in Case 2 of the Duke series, 5 craniotomies were necessary to prevent recurrent fixation of the bilateral skull morcellation. As pointed out by Ingraham and associates,[21] regrowth of bone is even more rapid in the infant's skull in which artificial bone defects have been made.

FIG. 6.—Linear craniectomy before application of polyethylene film.

In an effort to delay, or prevent indefinitely, regrowth of bone in linear craniectomy, Ingraham studied the reaction of inert foreign substances placed between the margins of artificially created channels in the experimental animal's skull. Of the group of substances studied, polyethylene film proved to have the most desirable physical properties. A report of its trial in 19 infants has confirmed the value of polyethylene in preventing rapid regrowth of bone in linear craniectomy.[20]

The technic of linear craniectomy with polyethylene film is a relatively simple one. In instances of complete synostosis of all suture lines, the procedure can easily be staged. Modern advances in pediatric anesthesia have controlled the major difficulties in this field, those early encountered by Lane. Fol-

lowing coronal, lambdoidal or parasagittal linear craniectomy, as indicated by the specific deformity to be relieved (fig. 6), a folded sheet of polyethylene

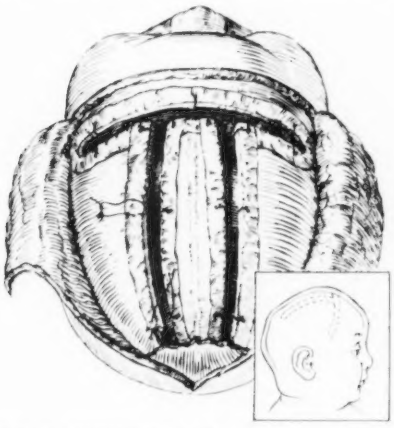


FIG. 7.—Diagrammatic sketch of linear craniectomy. The polyethylene film is sutured to the edges of the bony defects.

film is placed about each aspect of the craniectomy and sutured in place by black silk sutures placed through skull holes formed by modified Cone forceps (fig. 7). The procedure can be readily carried out and no postoperative complications have been noted that could be ascribed to the presence of this foreign body.

THE RESULT OF TREATMENT

Since our first report[11] of 5 cases in 1942 we have had an opportunity to study and treat an additional 27 instances of craniosynostosis. There were 21 males and 11 females. These patients could be clinically divided into three broad classes; there were 21 patients showing the changes characteristic of oxycephaly, 10 patients those of scaphocephaly and 1 of classical brachycephaly. Of particular interest to our present study is the fact that 19 of these patients presented abnormal ophthalmologic changes and that such changes were found only in the category of oxycephaly, or complete craniosynostosis. It is of further interest to state that no patient with scaphocephaly nor the single patient with brachycephaly showed visual changes. No changes in normal visual function or structure could be demonstrated in the age group below that of two years.

In order to classify the surgical problems that were encountered in this group, a further analysis in terms of age and visual findings upon admission for treatment is necessary. There were 16 of 30 patients first seen at an age above 2 years, including 8 between the ages of 2 and 10, 4 between the ages of 10 and 20, 2 in the age group of 20 to 30, and 4 who were 31, 32, 32 and 47 years of age respectively. The analysis made by Ingraham and associates[20] at the Children's Hospital describes but 5 cases of complete craniosynostosis, and no patient was studied for the first time at any age above that of 7.5 years. It is probable that many of Ingraham's patients were seen at such an early date that the classical picture of oxycephaly had not fully developed. It is evident that our series presented the opportunity to study the end result of untreated craniosynostosis and, in particular, changes in the visual apparatus, as well as unique surgical problems.

The abnormal visual findings in the 19 patients previously noted are listed as follows:

Exophthalmos	17
Optic atrophy	17 (one slight)
Papilledema	12
Divergent squint	8
Anisocoria	1
Nystagmus	1
Totally blind	2
Blind in one eye	3
Significant decrease in vision	9

The findings of severe grades of visual loss in 14 of these patients may be compared to Ingraham's observations in the younger age group.

On admission to the hospital, the two youngest patients showed definite papilledema bilaterally. Both of these showed complete remission of the papilledema after decompressive operation. The other three patients, all seen after they were 5 years of age, had various degrees of optic atrophy but no demonstrable loss of visual acuity. None of these had shown any progression of visual disturbance and it seems fair to assume that surgical procedures were effective in preventing any further loss of vision.

From a consideration of visual alterations, the end result in untreated craniosynostosis must be a variable one since patients have been seen, on the one hand, who are blind and, on the other hand, who show but mild exophthalmos and a divergent squint. The following abridged account is a fairly typical record of the visual changes in untreated oxycephaly.

J. E., present age 30, was first seen in the Medical Out-Patient Department in January, 1944, with complaints of sinus infection and visual difficulties. She stated that she was blind until the age of six, at which time she regained some vision in the right eye. She had had bitemporal headaches all her life. General medical and endocrine studies were normal. Of particular interest were the results of skull x-ray and eye studies. The skull roentgenogram showed absence of all suture lines, marked convolutional atrophy and a skull contour characteristic of oxycephaly. There was a pronounced exophthalmos with a divergent squint (fig. 8). Vision in the right eye was recorded as 20/300, in the left eye as 5/400. Bilateral optic atrophy was present. In November, 1951, vision was unchanged and an attempt was made to correct the marked right-sided exophthalmos by orbital and subtemporal decompression. One year later, there was minimal recession in the right-sided exophthalmos and visual acuity was recorded as 20/200 in the right eye and finger counting at three inches in the left eye.

Five of the 6 patients above the age of 20 years complained of intermittent generalized headache and in these patients, optic atrophy was manifest and visual loss was established and static. In this age group, a comparatively unusual one, perhaps relief of the chronically elevated intracranial pressure may best be obtained by subtemporal decompression.

FIG. 8.—Untreated instance of premature craniosynostosis. Case report is given in the text.

In the younger age groups, and in particular in our cases between the ages of 2 and 10 years, some form of linear craniotomy may not only stop the progress of visual loss but may indeed be followed by evidence of improvement in vision. An account of the latter instance has already been given (figs. 9 and 4A).

In 4 other patients in this age group, the further progress of visual loss has been prevented. Following operation, evidence for control of increased intracranial pressure may be demonstrated not only by regression of papilledema (fig. 9) but by increase in skull diameter and diminution in the degree of convolutional atrophy (fig. 10).

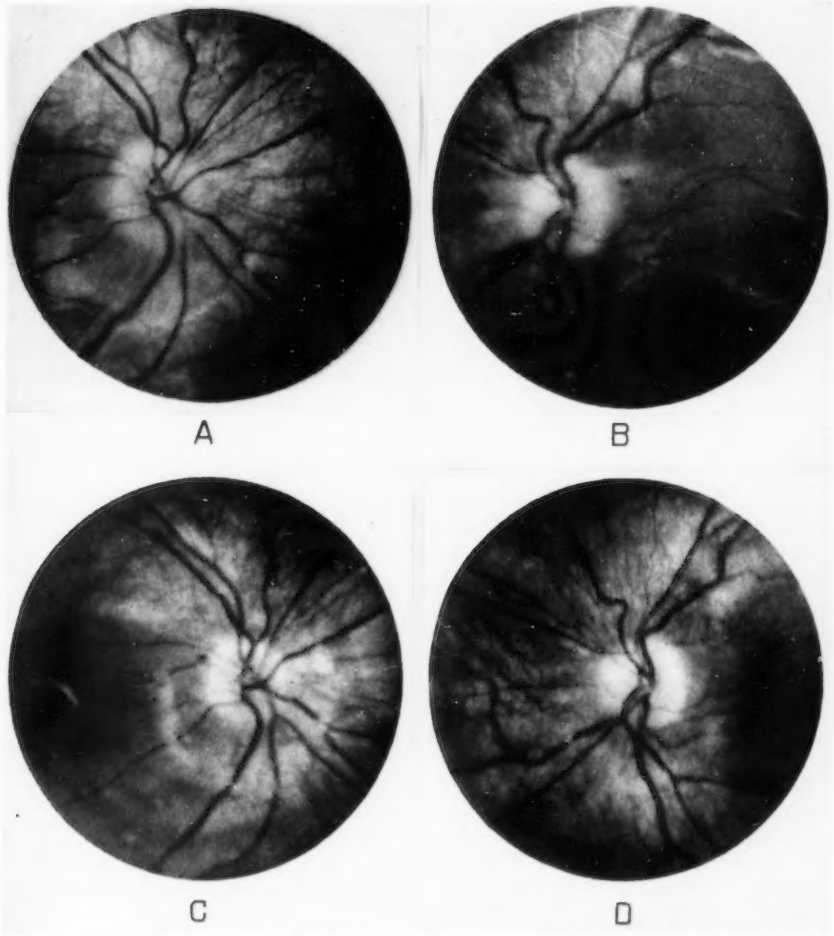


FIG. 9.—A and B, Preoperative papilledema (1948). C and D, Postoperative regression of papilledema (1952).

SUMMARY AND CONCLUSION

Various and often striking types of skull deformity may be caused by premature craniosynostosis. In one third or more of these deformities, usually in that designated as oxycephaly, optic atrophy subsequent to papilledema and diminution in visual acuity result. A review of the extensive literature devoted to this subject, and some of the case records studied in this report, suggest that these deformities are hereditarily conditioned. The visual disturbances are caused by an increase in intracranial pressure, secondary to the mechanical suppression of normal brain growth in the fused skull. Since brain growth is particularly active during the first two years of life, operative treatment should be instituted during this time to prevent

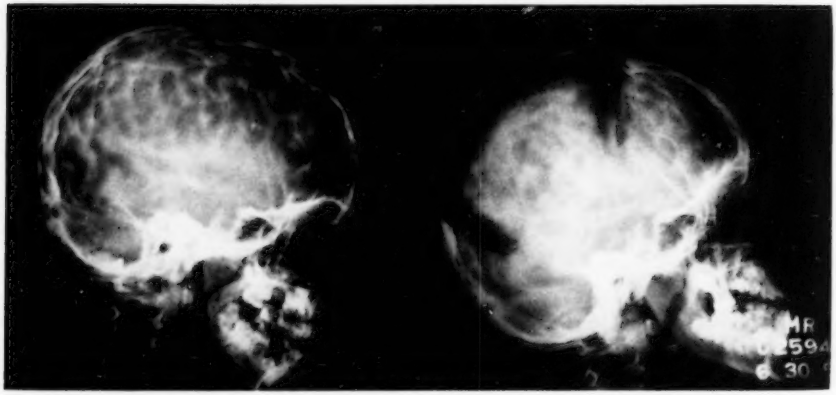


FIG. 10.—Characteristic skull roentgenogram of craniosynostosis of all suture lines. Post-operative view on right, illustrating change in convolutional atrophy after three years.

visual loss as well as to correct the skull deformity. Of the many technical procedures that have been advocated, linear craniectomy of or adjacent to the fused suture lines with the regrowth of bone controlled by the application of polyethylene film to the craniectomy line is the technic of choice. Even when craniosynostosis is recognized in the later years of childhood, marked improvement in vision may be accomplished by operative intervention or, in advanced instances of the deformity, further visual loss may be avoided. An opportunity has been afforded to study the end result of untreated oxycephaly in 6 patients over the age of 20 years. In 5 of these patients, visual loss was profound. The conclusion seems clear that the prevention of visual damage in patients with premature craniosynostosis is related chronologically to the recognition and treatment of the disorder.

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DISCUSSION

FORREST J. PINKERTON, M.D., Honolulu, Hawaii: Dr. Anderson and Dr. Woodhall have presented a thorough study and report of a condition which is not commonly observed in individual practice so far as it affects the vision. It has been pointed out that there are varying degrees of cranial deformity capable of producing ocular muscle and nerve abnormalities and visual defects, though they may not be so grossly apparent as the extremes we are discussing here.

I can add nothing to this excellent paper. I have had a couple of years to review the literature and I have come to the same conclusion as that reached by Dr. Anderson and Dr. Woodhall; that is, the nomenclature is so confusing even the experts cannot at this time unsnarl it.

I thought the audience might be interested in the 4 cases in one family that I have had under observation since 1938. With some difficulty I have been able to see them from time to time in follow-up. The two youngest children have been seen from birth. Their deformities were present in major degree since babyhood, and we report these cases not from the history but from our own observations.

We reported these cases in the *American Journal of Ophthalmology* in April 1952 and hope to follow up this first brief report from time to time. (Pictures shown by Dr. Pinkerton were in part the same as those printed in the *Journal*.) The mother presents all the characteristic signs. We can get no pertinent history of a similar condition in her family. She denies vigorously that anyone in her family has had any abnormality such as her own.

Literature reports that the nasal passages are not affected and Dr. Anderson in presenting this paper referred to normal nasal passages or usable nasal passages. Our experience does not confirm this, because all 4 affected cases in our group, including the mother, were mouth breathers, the nasal passages were narrow, the septum deformed, with high palatine arches and an excessive amount of adenoid tissue. I performed an adenoidectomy

on the two older children, and after harrowing anesthesia experience with both, I was astounded to find a large amount of adenoid tissue. These two patients are still mouth breathers, but they have less upper respiratory difficulty and their recurrent ear trouble has ceased.

Skull roentgenograms of all the family show small or absent frontal sinuses except for the father, and the maxillary sinuses in all are smaller than normal for their respective ages. The orbits are circular in shape, proportionately larger and shallow.

From our studies of the literature we would assume, since our cases were undoubtedly all born with the condition, that cerebral damage would be present, and that optic atrophy with progressive visual loss and mental deficiency would be present. However, we have not found this assumption entirely true. The 15-year-old boy may be slightly mentally retarded, and his best corrected vision is approximately 20/30 and 20/40. The youngest, a girl, has poor vision in the better eye and very poor vision in the other. This child has been unusually uncooperative. Practically all of our examinations on this 3½-year-old child have been done under general anesthesia.

The boy's fundus was normal, except for venous engorgement. We could not say that optic atrophy was noticeably present. This boy complains of headaches, but he had a marked refractive error, the correction of which improved his headache symptoms. The girl has definite pallor of the discs and an extreme right divergent strabismus. All three children and the mother had divergent squint, the smallest one being extreme, and all the family have refractive errors of moderate astigmatic or hyperopic type. The father, one son and one daughter were normal in all respects so far as their eyes and mentality were concerned.

Exophthalmometer readings of one patient, the 15-year-old boy, was 28.5 x 28 mm. The least reading was 27 mm. All had wide interpupillary measurements varying from 64 to 68 mm. except the youngest which could not be measured, but we would guess it to be even wider than the others. This child is unstable in walking. She walks in a rotary manner and has difficulty getting around chairs, yet she seems to be quite alert.

We have urged the family to permit surgery to the youngest child. They have refused even to discuss surgery. In the case of the oldest boy we have observed no progressive visual loss in the past twelve years and no progressive optic nerve changes; therefore, we have not urged surgery.

In recommending surgery we would be guided by five broad indications: (1) progressive exophthalmos; (2) progressive optic atrophy and/or diminishing visual acuity; (3) progressive mental deficiency, arrested or retarded mental development; (4) increased intracranial pressure with or without the above symptoms, and (5) cranial deformity with digital impressions and convolutional markings in the very young child before permanent visual loss and optic atrophy has occurred.

This last I consider very important, and I believe that it supports the opinion of Dr. Anderson and Dr. Woodhall.

Photographs show typical findings for all the cases except the severe tower skull shown by Dr. Anderson — the parrot beak, the exophthalmos, and the wide-set eyes. Roentgenograms show general similarity: cranial suture obliteration, prominent convolutional markings, dome-shaped skull, small paranasal sinuses, short-pointed nasal bones, large circular shallow orbital fossae, retardation of the facial bone development, and thinning of the skull walls, which was present in 3 cases. The mother did not appear to have such thinning of the skull walls, and this is the opinion of several roentgenologists who have studied these pictures.

OPHTHALMOPLEGIC MIGRAINE

A Discussion of Its Pathogenesis with a Report of the Pathologic Findings in a Case of Recurrent Oculomotor Palsy

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OPHTHALMOPLEGIC migraine was first defined by Charcot[7] in 1890 as a "condition associated with migraine in which there is a palsy of an ocular nerve, usually the third, temporary at first and later becoming permanent."

This clinical syndrome is characterized by paroxysmal, recurrent, transient partial or total ophthalmoplegia associated with severe hemicrania and with or without scintillating scotoma, homonymous hemianopsia, depression, general malaise, nausea and vasomotor disturbances. It frequently progresses to total and permanent unilateral oculomotor paralysis.

Numerous individual variations and atypical palsies have been adequately reviewed by Alpers and Yaskin[2] but the typical patient with ophthalmoplegic migraine presents the following clinical signs and symptoms.

1. There is a history of typical migraine attacks, without ocular muscle palsy, of many years' duration, prior to the first attack of ophthalmoplegia.

2. The individual attack may begin, as in any other migraine, with scintillating scotoma and homonymous hemianopsia followed by severe hemicrania and nausea and vomiting.

3. The headache and general malaise are usually much more severe and protracted in ophthalmoplegic migraine and may last from a few hours to many weeks. The headache is a true hemicrania.

4. The ophthalmoplegia may occur during or after the hemicrania and usually involves only the oculomotor nerve, although instances of trochlear and abducens paralysis have been observed.

5. When the third nerve is involved, it is the peripheral portion that is affected and invariably on the same side as the hemicrania. Rare instances of "alternating ophthalmoplegia" have been reported,[14] but in any given patient the paralysis almost always occurs on the same side.

6. The palsy becomes more severe with each successive attack until it is a permanent, unilateral, usually total, oculomotor ophthalmoplegia.

The reported high incidence of recurrent oculomotor paralysis associated with intracranial aneurysm, the long duration of the headache, the persistence of paralysis between attacks and the relatively lower percentage of familial migraine in these cases have led some observers[9] to suggest that ophthalmoplegic migraine is merely a recurrent

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oculomotor palsy with headache and not a form of migraine at all.

On the other hand, when one studies the case histories of a large number of these patients in whom an obvious migraine syndrome had been present for many years, later to be associated with recurrent oculomotor palsy without residual paralysis between attacks, one cannot escape the connection between the two syndromes.

Secondly, many migraine sufferers report diplopia occurring regularly during attacks. While manifest palsies are a somewhat uncommon occurrence in simple migraine, this diplopia is often indicative of minimal involvement of the oculomotor nerve.

Thirdly, the clinical symptoms and signs of both ophthalmoplegic migraine and simple migraine in their most typical form have been produced by organic intracranial pathology such as brain tumor, subdural hematoma and intracranial aneurysm. In these cases, however, the initial transient symptoms rapidly progressed to irreversible signs, the temporary ophthalmoplegia became a permanent oculomotor palsy and the scintillating scotoma became a homonymous hemianopsia.

Lastly, a comprehensive review of over 1,600 cases of migraine, culled by Donahue[8] from the literature, demonstrated that various ocular palsies occurred in 17 per cent. In most instances the paralysis lasted for a period of three to four days, slowly passing away but occasionally persisting for weeks or longer. In some recorded cases recovery became less perfect as the attacks persisted, and finally the paralysis became permanent (in one case in the absence of further headache). Diplopia was reported in 14 per cent of cases; third nerve involvement occurred in 88 per cent of cases with muscle palsies. Paralysis of ocular muscles was recorded in 296 cases with no case of complete bilateral paralysis or bilateral ptosis. Unilateral dilated and fixed pupil was seen in 76 cases. A unilateral dilated pupil with preservation of light reaction was noted in 49 additional cases and inequality of the pupils was recorded in 423 cases.

When the etiologic factor in the production of the symptoms of both simple and ophthalmoplegic migraine is physiologic or vasomotor in character, the reversibility of the process is easily understood and the difference between the two syndromes is largely a question of severity and degree.

Classic migraine, also variously known as ophthalmic migraine, simple migraine and migraine, has been defined by Riley[25] as "a periodic incapacitating headache culminating in nausea or vomiting, often preceded by visual disturbances, followed by sleep and occurring against the background of relatively perfect health."

While this definition may fit the picture of the "typical case" there is such variety in the symptom complex of migraine as to preclude a true clinical symptomatology. Cases of unquestioned migraine are seen in which the headache is missing from the symptom complex but in which the scintillating scotoma, homonymous hemianopsia and nausea leave no doubt as to the diagnosis. Any single symptom or combination of symptoms and signs may appear in such a way as to firmly establish the nature of the disturbance and yet have little resemblance to the "classic migraine attack." Likewise the degree and severity of the attack may vary within such wide limits as to be hardly noticeable on the one hand to complete prostration of the victim on the other. About the only thing common to most cases of migraine is the personality pattern underlying the condition; a pattern so characteristic as to be of utmost importance in both diag-

nosis and treatment and ably described by Alvarez[3] and numerous other authors, including Wolff.[28]

Wolff's concept[28] of the pathogenesis of migraine is now generally accepted. He demonstrated that the preheadache phenomena of migraine (aura, irritability, scintillating scotoma and hemianopsia) are due to vasoconstriction of cerebral arteries and that the headache is brought about by vasodilation of vessels of the scalp and dura, primarily branches of the external carotid artery.

When dilatation of cranial arteries is prolonged over several hours, a transient thickening of their walls results and this may have a definite effect in producing unilateral intracranial volume changes of considerable degree.

The intracranial volume changes from one phase of a migraine attack to the next have been vividly demonstrated by Goltman[15] in a patient with a left frontal bone defect.

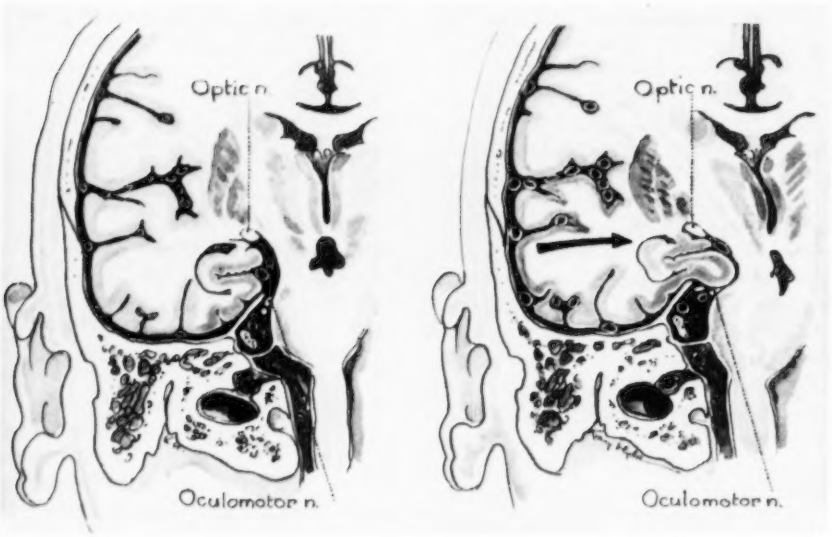
This patient had had migraine attacks since childhood. Immediately preceding the headache the patient's face became blanched and there was a definite depression at the site of the bone defect as compared with its usual state. As the headache developed, the skull depression filled until ultimately the intracranial contents protruded, assuming the appearance of a tumor. At this point the patient began to vomit and the face became flushed. With the subsidence of the headache in twelve to seventy-two hours the intracranial contents resumed their former relation to the bone defect.

Naffziger[22] was able to show these intracranial volume changes in migrainous patients in a different way. In two individuals with a long history of typical migraine he demonstrated by means of roentgenograms taken before and during the headache phase of a migraine attack, a lateral shift of the calcified pineal gland away from the side of the hemicrania.

The pathogenesis of ophthalmoplegic migraine is unknown. The prevailing theories are largely vascular in concept but much confusion exists and the five cases clinically diagnosed as ophthalmoplegic migraine which have come to autopsy have shed little light on the problem. Weiss[27] found warty granulations on the left oculomotor nerve and over the base of the brain in which tubercle bacilli were found. Richter[24] described a fibrochondroma involving the right oculomotor nerve. Karplus[19] reported a parenchymatous syphilis of the brain with a neurofibroma of the right oculomotor nerve. Shionoya's case[26] was one of tuberculous meningitis with enlargement of the right oculomotor nerve due to connective tissue proliferation. Alpers and Yaskin[2] found nothing at autopsy which would account for oculomotor paralysis.

In the main, theories as to the mechanism of production of ophthalmoplegic migraine are postulated on pressure on the oculomotor nerve from dilation of the large vessels which are in close anatomic relation to the nerve. Bramwell[5] considered the paralysis as due to pressure upon the nerve either by a leaking aneurysm, an intact aneurysm or an artery with an abnormal relationship to the nerve. Symptoms resulted when the vessel or the aneurysmal sac was temporarily distended by vasomotor changes such as occur in an attack of migraine.

Ehlers[10] pointed to the posterior cerebral artery as the anatomic basis of a functional connection between the occipital cortex and the homolateral oculomotor nerve. The posterior cerebral artery, running in intimate relation to the third nerve is the sole vascular supply of the visual area in the calcarine cortex, and he suggested that vascular changes in the occipital lobe are the cause of the various visual phenomena associated with simple migraine while pressure of the same vessel on the third



a b

FIG. 1.—Cut section through the brain. a, Normal relationships of the hippocampal gyrus to the tentorium and the oculomotor nerve. b, Hippocampal gyrus herniation over the free edge of the tentorium with pressure on the third nerve.

nerve would account for the paralysis seen in ophthalmoplegic migraine.

We have recently studied a case in which typical hemicrania and transient homonymous scintillating scotoma were followed by the development of a permanent homonymous hemianopsia, which was found to be due to a leaking aneurysm of the anterior communicating artery, demonstrated by arteriography and later by intracranial exploration. The site of the pathology was far distant from the vascular supply of the calcarine fissure and in no way related to the oculomotor nerve. There was transient, very slight dilation of the homolateral pupil, associated with increased intracranial pressure which was relieved by clipping the vessel.

Angioneurotic edema, with or without transitory hypophyseal swelling, has been suggested as a cause of this condition by Elliot,[11] and Fry[14] has recently reported a case of recurrent oculomotor palsy associated with angioneurotic edema.

All of these theories, however, are in conflict with the careful studies of Wolff[28] and his co-workers who, as previously mentioned, have shown that migraine headache is due to distention and stretching of the branches of the external carotid artery, none of which are in close proximity to the oculomotor nerve.

For a number of years it has been well known that herniation of the hippocampal gyrus of the temporal lobe over the free edge of the tentorium, due to an ipsilateral expanding cerebral lesion, can produce pressure on the oculomotor nerve as it crosses the body of the sphenoid bone (fig. 1). This pressure may be sufficient to paralyze the nerve partially or completely. Hippocampal gyrus herniation has not, heretofore, been suggested as a factor in the patho-

genesis of recurrent oculomotor paralysis because recurrence of the paralysis has not been a prominent feature of the herniation.

Meyer,[20] in 1920, first described temporal herniation through the incisure of the tentorium and pointed out that the uncus is the portion of the brain which, from its position, is most liable to herniate (fig. 2). Since then reports of numerous cases have testified to the localizing value of unilateral dilation of the pupil in cases of extradural hemorrhage, tumor, abscess or any unilateral intracranial expanding lesion. Autopsy reports have amply demonstrated that this pupillary dilation is produced by pressure of the herniated uncus on the homolateral third nerve.

FIG. 2.—Normal relationship of the upper edge of the tentorium to the uncus.

Holman and Scott[17] and Cairns[6] have emphasized the occasional *transient* character of this pupillary reaction. Woodhall and associates[29] state that "a dilated and fixed pupil, either of permanent or *transient* nature remains the most valuable lateralizing sign and may, in many instances be caused by herniation of the corresponding hippocampal gyrus."

The work of Reid and Cone[23] is most convincing in this regard. They have observed such herniation in head injuries and noted its relation to homolateral dilation of the pupil. They have confirmed the mechanism of its production by experimental work on monkeys and have proved beyond doubt the relationship of unilateral increased intracranial pressure to hippocampal gyrus herniation and peripheral third nerve palsy. A description of their experiments clearly demonstrates the *reversibility* of the process.

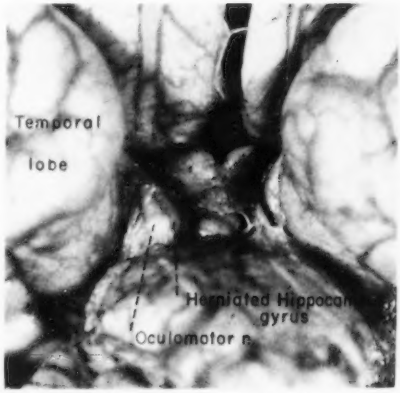


FIG. 3.—Ventral surface of the brain showing herniated hippocampal gyrus exerting pressure on the oculomotor nerve.

> The extradural compression in the experimental acute herniations was obtained through the use of a Forbes window screwed tightly into a trephine hole in the skull, the position of which varied in different animals between the frontal, parietal and temporal regions. After the dura had been separated from the calvarium for a short distance around the trephine hole, Ringer's solution was allowed to flow in and the pressure exerted could be altered and recorded at all times. . . .
>
> It was possible to produce at will an ipsilateral pupillary dilation and to watch the pupil return to normal on release of the pressure. After the pressure had been kept at a level sufficiently high to hold the pupil in complete dilatation and fixation, the animals were killed. Ipsilateral hippocampal herniation was found as a routine at necropsy. The herniation was great in the animals showing marked pupillary change and less extreme in those showing lesser degrees of involvement of the third nerve.

Jefferson's description[18] of the anatomic relationships of the tentorium is of special interest:

No perfect illustration of the tentorium seems to exist—none that gives a correct impression of its anteroposteriorly convex upper surface, its lateral decline and the manner of the abrupt falling away of the almost vertically directed anterior 2 cm. of its surface as it changes its architecture to sweep forward onto the sides of the cavernous sinus. The temporal lobes lie on the tentorium which slopes away laterally as a gently inclined plane, so that pressure from above will tend to make them slide away from the midline. However, if one lobe is enlarged it cannot escape overhanging the free edge. For this reason, a tumor of the temporal lobe will be the surest way of bringing it more firmly into contact with the midbrain and squeezing its inner border over the sharp edge of the falx, into a situation in which it can herniate downward into the posterior fossa. . . . There is no doubt that if the tentorium were horizontal, herniation would be more common than it is. Its tentlike form causes it to protect the midbrain.

It is clear that hippocampal gyrus herniation is not simply the result of generalized increase in intracranial pressure, which more often produces paralysis of the sixth nerve. Only the unilateral feature of the process can explain the localizing value of homolateral pupillary dilation in head injury, tumor and the like.

From clinical, pathologic, anatomic and experimental evidence it is plain that herniation of the hippocampal gyrus is due to relative increase in pressure in the ipsilateral half of the cranial cavity and that it is often a *transient* and *reversible* process. The idea that uncal herniation is catastrophic and irreversible has arisen because the condition is most frequently seen at autopsy in cases with sustained high intracranial pressure.

The following case report with autopsy findings of marked herniation of the hippocampal gyrus of the temporal lobe, producing recurrent oculomotor paralysis, suggests the pathogenesis of ophthalmoplegic migraine.

CASE REPORT

L. D. S., age 50, was admitted to the neurosurgical service of the Veterans Hospital at Fort Miley, San Francisco on November 6, 1951. He complained of generalized headache with severe paroxysmal exacerbations associated with difficulty in concentration. He had lost 50 pounds in six months. During the preceding three weeks he had noted periodic dizziness, most marked on change of position. He had also noted a periodic loss of urinary sphincter control, a peculiar taste to all his food and marked diminution in his sense of smell. The patient had noted diplopia on one occasion, and his brother had noted a divergent strabismus associated with an attack of extreme head pain. The patient's family had noticed, between attacks of headache, that he had seemed much improved both physically and mentally.

Physical examination was entirely normal except for the following: There was a bilateral partial anosmia, more severe on the left. The right pupil was slightly larger than the left (subsequent examination revealed pupils which were equal) but both reacted well to light and accommodation. Visual acuity was 20/20 in each eye. Visual fields were normal for multiple isopters. There was a bilateral papilledema of two diopters elevation with a few retinal hemorrhages near the discs. The tongue deviated slightly to the left and a left facial hypalgesia and left masseter muscle weakness were noted. Right-sided hypalgesia of an indefinite nature and equivocal bilateral Babinski reflexes were recorded.

Roentgenograms of the skull showed an irregular thinning of the inner table, sharply circumscribed in both frontal areas, presumably due to enlarged diploic vessels.

Chest roentgenograms revealed a hemisphere of soft tissue density in the right upper lung field which was sharply circumscribed and suggested bronchogenic carcinoma.

Electroencephalogram was reported as a diffusely abnormal record with a question of focus in the right temporal region, mostly anterior.

Lumbar puncture revealed a pressure of "over 600 mm. water" with 40 cells per cubic millimeter, 90 per cent of which were red blood cells.

The patient had six known attacks of transient rigat third nerve palsy, each associated with paroxysmal exacerbation of a right frontal headache occurring over a period of about sixteen days. The first attack was observed as a transient diplopia by the patient himself. The second attack was observed as a temporary divergent strabismus by the patient's brother. The last four attacks were observed by us from onset to conclusion and presented the typical clinical picture of ophthalmoplegic migraine as follows:

The patient was brought to the eye department on November 6 in a horizontal position on a gurney. He was not complaining of discomfort or other symptoms but on questioning admitted to a slight generalized headache. He was alert, well oriented, cooperative, was able to walk well and did not look particularly ill. Shortly after sitting up in a chair for purposes of visual field study he began to complain of terrific right-sided headache and was obviously in extreme distress. From being alert and cooperative he rapidly became very inattentive and would not respond except to loudly and sharply spoken command, and then very slowly. His face became pale and he perspired profusely. At the same time he complained of intense urgency to urinate and, in fact, did lose urinary sphincter control. These prodromal symptoms had lasted about three minutes when it was noted that his right eye was beginning to diverge and the patient admitted to diplopia when questioned. Within the next thirty seconds he developed a complete paralysis of the right internal, superior and inferior rectus muscles and the right inferior oblique as well as a widely dilated right pupil which was fixed to light and accommodation. At the end of another two minutes a paralysis of the levator muscle of the right lid had developed with resulting complete ptosis. The patient was nauseated and groaning with pain and was returned to his horizontal position on the gurney, where he lay on his right side. Within five minutes the hemicrania had disappeared, the patient was again alert and cooperative with normal skin color and absence of perspiration. Examination revealed a complete recovery from the oculomotor paralysis with normal and equal lid apertures, normal ocular rotations and normal pupillary size and reactions.

Three additional, practically identical, attacks were observed. Each appeared to be precipitated by changes in the patient's position.

On November 8, 1951 the fourth attack of ophthalmoplegic migraine was witnessed but this attack did not reverse itself. The headache continued and the ophthalmoplegia remained as a permanent and complete oculomotor palsy until the patient's death on November 13, 1951.

The patient grew rapidly worse after November 8 and because of the possibility that the oculomotor palsy was due to an intracranial aneurysm, arteriography was performed on November 13, 1951. The patient died that evening.

In discussing this case with Dr. Edwin Boldrey[1] we were referred to the work of Reid and Cone[20] as a possible explanation of the oculomotor palsy.

The arteriograms demonstrated a large right frontal lobe tumor with a smaller temporal lobe lesion. There was a shift to the left of the right middle and anterior cerebral arteries.

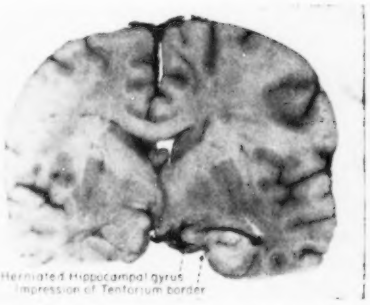


FIG. 4—Cut section of the brain showing herniation of the uncus with indentation of the brain by the incisure of the tentorium.

Autopsy revealed a bronchogenic carcinoma of the lung with cerebral metastasis. In the right frontal lobe, 1 cm. from the tip and 2 cm. from the midline there was a hard, round, heavy, solid tumor which shelled out completely and measured 3 cm. in diameter. There was marked edema of the brain posterior to the tumor. In the right parietal area a similar tumor was found 2 cm. from the midline and 7 cm. from the tip of the occipital lobe.

Examination of the uncut brain revealed a marked herniation of the right hippocampal gyrus over the edge of the tentorium with pressure on and stretching of the right third nerve (fig. 3). Further studies of the cut brain revealed the extent of the herniation and the degree of pressure on the oculomotor nerve (fig. 4). As the probable cause of death there were many hemorrhages in the brain stem (Jefferson [18]).

COMMENT

Recurrent oculomotor paralysis with the typical clinical picture of ophthalmoplegic migraine was here produced by herniation of the hippocampal gyrus of the temporal lobe over the free edge of the tentorium, with pressure on the oculomotor nerve. The uncal herniation was secondary to unilateral cerebral edema with changes in brain volume such as to force the uncus through the incisure of the tentorium and onto the third nerve.

In this case the recurrent and reversible oculomotor paralysis did not occur as "a syndrome of vascular origin."[10] It was not due to "vasomotor phenomena."[5] It was not the result of a "leaking or intact aneurysm."[5] But in so far as any of these is capable of producing a unilateral increase in cerebral volume they could have produced the same result as was here produced by brain tumor.

From the purely anatomic point of view it is not difficult to explain the mechanism of production of hippocampal gyrus herniation and, conversely, the reversibility of the process. The anatomic relationships of the structure have been succinctly described by Jefferson,[18] as previously noted. Just as variations in the degree of unilateral increase in cerebral volume would account for temporal herniation occurring in some cases and not in others, so also anatomic variations in the slope of the tentorium and the size of the hiatus might account for greater ease of herniation in some individuals than others (fig. 5). The more horizontal the tentorium and the larger the hiatus, the less force would be required to produce the brain hernia. Conversely, the anatomy of the tentorium is such that a herniation of the gyrus would strongly tend to right itself if the pressure on it were relieved, reversing the symptomatology produced by the hernia.

These anatomic variations might well explain the fact that while some subdural hematomas, some temporal lobe tumors and even some frontal lobe tumors produce uncal herniation, most do not. In some individuals migraine headache is repeatedly followed by oculomotor palsy, whereas in most cases ophthalmoplegia never occurs.

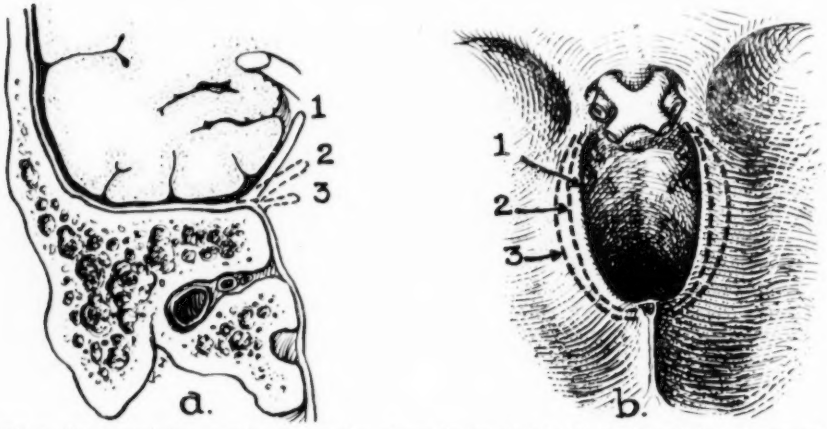


FIG. 5.—Exaggerated diagrammatic representation of possible variations in (a) the slope of the tentorium and (b) the size of the hiatus.

In this regard the most recent case of Alpers and Yaskin[2] is of interest to us.

The patient exhibited typical clinical ophthalmoplegic migraine with recurrent left oculomotor palsy and headache. Intracranial aneurysm was suspected, and arteriography of the left internal carotid artery and exploratory craniotomy were performed, following which the patient

became comatose and died. Autopsy revealed thrombosis of the internal carotid artery, an extradural hematoma, herniation beneath the falx of the medial surface of the left hemisphere and herniation of the left hippocampal gyrus. No aneurysm or vascular anomaly was found to account for the ophthalmoplegic migraine.

Even if we believe that, in this case, the herniation was secondary to the unilateral cerebral edema produced by the thrombosis of the internal carotid artery and the subdural hematoma, it is interesting to note that here was an individual in whom temporal herniation was apt to occur and that there is no other explanation for the preoperative existence of typical ophthalmoplegic migraine of many years' duration.

The role of uncal herniation, with or without ophthalmoplegia, is important in explaining certain other phenomena associated with migraine.

When one considers that the ocular muscle plasies reported by Donahue,[8] and noted above, were in cases of ordinary migraine and not ophthalmoplegic migraine, the very high incidence of involvement of the ocular musculature (especially that under control of the oculomotor nerve) is indeed striking and indicates a much greater frequency of this phenomenon in simple migraine than has hitherto been suspected. No other mechanism would explain these findings as well as does the fact of temporal lobe herniation of varying degree.

The occurrence of contralateral transient hemiplegia and paresthesias of varying degree in association with attacks of simple or classic migraine is well known and can best be explained by compression of the crus and pyramidal tracts by herniation of the brain through the incisure of the tentorium.

The pain of ophthalmoplegic migraine is generally described as quite different from ordinary migraine. It is more severe, usually of longer duration, often precedes or coincides with ocular palsy, is definitely localizable, but trigeminal in distribution and could easily be accounted for by the complications of hippocampal gyrus herniation. It is probably due to the pressure of the herniated uncus on the branches of the trigeminal nerve which richly supply the area below it.

Visual defects are frequently associated with both classic and ophthalmoplegic migraine. In the former the so-called scintillating scotoma is the predominant symptom, occurring in a high percentage of cases and often merging into transient homonymous hemianopsia which is succeeded by hemicrania.

For the most part the visual defects are transient and even evanescent in character. The scintillating scotomas are usually clearly homonymous and often described as crude, half-formed and bizarre visual hallucinations. The homonymous hemianopsia is usually quite complete, but not dense in character and lasts for twenty or thirty minutes. It is Wolff's view[28] that the preheadache visual phenomena of migraine are due to cranial vasoconstriction and are localizable in the visual cortex in the occipital lobe. They result from occlusion of the branches of the posterior cerebral arteries, causing ischemia of the most peripheral portions of the artery's blood supply, that area near the occipital pole, and spread ultimately to involve the anterior portions of the calcarine fissure and hence the peripheral portion of the visual field. Wolff infers, as previously remarked, that the preheadache visual phenomena result from dysfunction of cerebral vessels in contrast to the headache phenomena which result chiefly from dilation of extracerebral vessels.

Besides Wolff's experiments there is considerable additional evidence for this hypothesis in the case reports of Adie[1] and the electroencephalographic studies

of Engle and his co-workers.[12,13] These observers found that electrical disturbances, similar to those seen during cerebral ischemia, could be obtained from the electrodes over the occipital cortex during the scotomatous phase of migraine, both spontaneous and induced by decompression sickness.

In the first description of hippocampal gyrus herniation by Meyer[20] he described the strangulation of the posterior cerebral artery by the edge of the tentorium and the "collapse of the corresponding part of the occipito-temporal cortex." He seemed to be particularly concerned with the possibility of hemianopsia as a false localizing sign from obliteration of the posterior cerebral artery. Greenfield[16] and Moore and Stern[21] were also concerned with the obliteration of the blood supply to the area striata in cases demonstrating brain herniation into the hiatus of the tentorium. Reid and Cone[23] reported autopsy findings of ipsilateral occipital lobe infarction in three out of six cases of hippocampal gyrus herniation with dilated pupil.

In his description of the tentorium, Jefferson[18] dwells specifically on this point:

> Sloping obliquely backward on either side and partly encircling the midbrain lie the posterior cerebral arteries, and it is a matter for surprise that they do not more often suffer from their close relationship to the sharp edge of the tentorium. No other intracranial vessel is vulnerable in this manner in any thing approaching the same degree.

It would appear from the above that the visual disturbances associated with ophthalmoplegic migraine may be explained on the basis of occipital lobe ischemia, or even infarction, produced by compression of the posterior cerebral artery between the sharp edge of the tentorium and a herniated hippocampal gyrus.

The visual phenomena of migraine are more transient because the physiologic, vasomotor disturbances which produce the arterial dysfunction are more fleeting, though even here permanent damage may occur.[1]

The visual field loss in ophthalmoplegic migraine usually also starts as a transient and reversible sign but may become permanent, just as the initially reversible oculomotor palsy is finally made fixed.

From the observations of Naffziger,[22] Goltman,[15] and Wolff[28] the intracranial volume changes occurring in migraine are known. These changes, physiologic or vasomotor in origin, are considered by us to be capable of producing, *in susceptible individuals*, a hippocampal gyrus herniation of a degree sufficient to compress the oculomotor nerve and related structures. The transient character and reversibility of the symptoms are correlated with their etiology and are in direct proportion to the degree, duration and cause of the unilateral increase in cerebral volume producing them. Thus, a phenomenon of vasomotor origin would be expected to recede with the subsidence of the "vegetative storm" which brought it about. With repeated and prolonged "attacks" certain relatively irreversible changes in the insulted tissue would eventuate so that each new change would be added to the residuum of its predecessor until a permanent loss of function ensued.

When the cerebral edema causative of the uncal herniation is due to organic pathology such as tumor or hemorrhage, the resulting clinical picture is apt to be more violent in its manifestations, with transient characteristics absent or quickly merging into permanent change. But regardless of the etiology of the unilateral increase in cerebral volume, it is the temporal lobe herniation which is

responsible for the clinical picture of ophthalmoplegic migraine in one or another of its protean forms.

SUMMARY

1. Charcot's original definition of ophthalmoplegic migraine as "migraine associated with palsy of an ocular nerve" is still valid. The two clinical entities may be strikingly similar and classic migraine frequently merges into the ophthalmoplegic type.

2. The various theories as to the pathogenesis of ophthalmoplegic migraine are, to date, largely pathologic in concept. They have little in common with accepted ideas on the etiology of migraine.

3. Anatomic, clinical, pathologic and experimental evidence have been cited to prove that a unilateral increase in cerebral volume can produce ipsilateral herniation of the hippocampal gyrus of the temporal lobe through the incisure of the tentorium, resulting in recurrent homolateral oculomotor palsy, visual disturbances and hemicrania.

4. A case of recurrent oculomotor paralysis is reported, presenting the typical clinical symptom complex of ophthalmoplegic migraine. Autopsy findings revealed a marked herniation of the hippocampal gyrus with pressure on the third nerve. The hernia was secondary to unilateral cerebral edema produced by a brain tumor.

5. A new theory of the pathogenesis of ophthalmoplegic migraine is advanced. It is suggested that the symptom complex is produced by hippocampal gyrus herniation *in susceptible individuals;* that the herniation of the brain over the free edge of the tentorium is dependent on anatomic variations correlated with unilateral cerebral edema, and that the edema may be the result of intracranial volume changes of physiologic or pathologic etiology.

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DISCUSSION

FRANK B. WALSH, M.D., Baltimore, Md.: Dr. Harrington and Dr. Flocks have stated clearly what seems a reasonable deduction concerning a determining factor for the development of ophthalmoplegia in migraine. The autopsy finding of cerebral herniation producing pressure on the third nerve strongly supports their thesis. The paper which we have just heard promises to be a significant contribution. Further studies are in order. In arteriography we have a new and illuminating method of study which may be particularly useful in these cases.

There are many clinicians, and I am one, who for years have believed that ophthalmic and ophthalmoplegic migraine are closely related disorders or variations of the same disorder. We have observed individuals who after suffering from ophthalmic migraine for years suddenly develop ophthalmoplegia.

I am confident that the essayists would agree that variations in the anatomic relationships in different individuals may at least in part control the determination of the principal clinical features of migraine. The anatomy which seems to underlie the development of ophthalmoplegia may be equally important as regards aphasia, hemianopsia and the like. The area including the edge of the tentorium, third nerve, overlying cerebrum and the posterior cerebral artery is vital to the proposition which has been stated as regards ophthalmoplegia. Obviously changes in the position or size of the posterior cerebral artery might influence the third nerve. I wish to speculate briefly on the posterior cerebral artery in migraine only as regards partial hemianopsia, which occasionally is persistent.

On the basis of the blood supply to the optic radiations it would seem that in any cases characterized by partial hemianopsia there would be a tendency toward sparing of the lower quadrants of the defective half-fields. In looking over my material I have found that such a permanent loss of homonymous upper quadrants had been charted in four of five such cases. It is of some interest that such a tendency toward permanent defect in the upper quadrants is precisely the opposite to what is found in thrombosis of the internal carotid artery when there is an associated hemianopsia. In it there is a tendency toward sparing of the upper quadrants of the defective half-fields.

It is possible that persistent hemianopic field defects in migraine may be due to pressure on a prolapsed temporal lobe or to interference with venous drainage. In any event, it is of some interest that permanent field defects have been located in the upper half-fields in a majority of the cases which I have observed.

HUGH C. DONAHUE, M.D., Boston, Mass.: Episodes of migraine are occasionally associated with more or less pronounced pareses of certain cranial nerves, more often the oculomotor, which may be transitory in nature or may persist for longer periods. The term ophthalmoplegic migraine has been subjected to a multiplicity of interpretations in the literature, often being alluded to as any ocular palsy which has been accompanied by headache. This is far from its strict and literal meaning, which refers actually to a palsy of one or more ocular muscles in the presence of an attack of true migraine; as the etiology of true migraine is far from settled, obviously the etiology of ophthalmoplegic migraine cannot yet be specifically determined, but certain suppositions can be made, dependent upon our present

knowledge of the mechanisms of symptom production of the migraine complex.

Dr. Harrington has very interestingly reported the clinical course and autopsy findings of a case of bronchogenic carcinoma metastasizing to the brain which was accompanied by generalized headache and increased intracranial pressure. The increased intracranial pressure caused herniation of the uncus or hippocampal gyrus with the resultant pressure upon the third cranial nerve causing ocular muscle paralysis. The reported case has been augmented by some very beautiful illustrations; certainly no one would disagree with the findings in this case of intracranial tumor. Similar cases have been seen upon the neurologic service of Dr. D. Denny-Brown and Dr. Joseph Foley of the Boston City Hospital and have been investigated clinically and post mortem. They have found that such a syndrome is not uncommonly produced by such lesions as ruptured aneurysms, metastatic tumor such as glioblastoma multiforme with pontine hemorrhage, cerebral infarct and cerebral hemorrhage accompanied by brain edema. The usual supratentorial lesions, however, which may cause herniation of the uncus through the tentorium with resulting compression of the midbrain are accompanied by fever, rapid pulse and respiration, dilatation of one or both pupils, complete third nerve palsy, disorder of awareness or impairment of consciousness. In every case studied at this neurologic unit these symptoms were present. In true ophthalmoplegic migraine, however, such constitutional disorders are not usually present, as the patient, although sometimes prostrated and incapacitated, never loses consciousness or exhibits symptoms indicative of coma. Having in mind the production of ocular palsy by the above-mentioned method, Dr. Harrington then postulates that in "susceptible individuals," in whom anatomic variations may be present tending toward more facile production of uncal herniation and in whom cerebral edema of varying intensity may occur as a result of physiologic or vasomotor phenomena, pressure upon the oculomotor nerve may be exerted and ocular palsy may result; further that such conditions may be reversible and are in direct proportion to the "degree, duration and cause of the unilateral increase in cerebral volume producing them." Surely such conditions as described cannot be gainsaid, but on the other hand, in the light of present evidence must be deemed quite conjectural and gratuitous. It is not certain that transitory changes in intracranial volume can produce herniation of the uncus, which is quite a substantial mass of tissue.

Experimentation upon dogs performed at the Boston City Hospital neurologic unit has demonstrated that both in these animals and during certain surgical procedures in humans, upon dilatation of the cerebral vessels with carbon dioxide, such vessels being visible by exposure of the convexity of the brain and productive of definite cerebral hyperemia, no herniation of the hippocampal gyrus resulted. So it is debatable whether vasomotor phenomena can produce a sufficient degree of increased intracranial pressure to cause hippocampal gyrus herniation.

In a series of cases of migraine both with and without ophthalmoplegia, the dynamics and chemical properties of the cerebrospinal fluid were studied by Merritt and Von Storch; in no instance was the cerebrospinal fluid pressure elevated, which would seem to be a necessary factor in the production of herniation of the uncus.

The production of third nerve palsy in the presence of true migraine obviously may have other possibilities. Indeed, it would appear that because of the recognized production of migraine symptoms by vascular changes, associated ocular palsies would also be more logically explained by abnormalities occurring in the cerebral vessels anatomically adjacent to the third, fourth or sixth nerve; it is not necessary to review the anatomy which has been done by various authors, notably Sunderland in 1948, but merely to recall that the oculomotor nerve in its course is closely associated with the basilar, posterior cerebral, posterior communicating, superior cerebellar and internal carotid arteries and that it may very well be affected by pressure from small congenital aneurysms during the phase of migraine vasodilation or leaking aneurysms which result in thickening of the wall of the lesion. Aneurysms of the carotid artery are occasionally difficult to demonstrate by internal carotid arteriography. Those on the more posterior vessels never can be visualized by internal carotid injection; rather their demonstration requires vertebral angiography. It seems quite possible, however, that such lesions could account for transitory ocular pareses, and a leaking aneurysm with thickened media producing long-continued pressure upon the nerve might also account for more permanent ocular pareses.

It must be admitted that the actual cause of ocular pareses occurring with true migraine is still far from being specifically determined. I wish to congratulate Dr. Harrington, who has brought forward an interesting and fascinating theory as to their production; however, in formulating a diagnostic scheme for handling cases exhibiting symptoms of this entity, I would suggest (1) a complete neurologic examination; (2) flat x-ray films of the skull, particularly of the base since vascular lesions are commonly visible by flat x-ray; (3) internal carotid arteriography to demonstrate lesions of the internal carotid artery and (4) vertebral arteriography to demonstrate the more posterior vascular lesions.

It is through the gradual and progressive evolution of studies such as those made by Dr. Harrington that the true nature of the condition causing ophthalmoplegic migraine will be disclosed. In the light of our present knowledge, however, of migraine symptom production being generally attributed to vascular abnormalities it would seem to me that accompanying ocular muscle changes would also be more plausibly explained upon the basis of pathology in the vascular branches located adjacent to the affected nerve and exerting pressure upon it.

DR. HARRINGTON: May I thank both Dr. Walsh and Dr. Donahue for their very complete discussions and their considerable contribution to the subject of the pathogenesis of opthalmoplegic migraine?

I have nothing further to add except to say that without numerous careful autopsies, it is quite obvious that one cannot as yet present proof of the etiology of ophthalmoplegic migraine. It is equally true that the theory which we have advanced as to its pathogenesis is, in part, speculative. This speculation, however, is founded on good anatomic, clinical, and pathologic evidence, and while it is subject to confirmation and further proof, it is equally subject to disproof. It is hoped that our speculation, based on observed pathology, will stimulate others to more careful postmortem observations in numerous cases of this type, thus affording more concrete proof of this theory of the pathogenesis of ophthalmoplegic migraine.

FURTHER OBSERVATIONS ON THE SYNDROME OF GLAUCOMATOCYCLITIC CRISES

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OUT OF the bewildering variety of manifestations which are included under the glaucomas, one group of cases stands out as having so many characteristics in common that anyone who has encountered one case will immediately recognize any other case belonging to this clinical entity. This is a syndrome of recurrent attacks of acute noncongestive glaucoma, usually associated with signs of mild cyclitis, which follows a similar episodic pattern.

In 1948, we reported nine cases belonging to this group.[5] We showed that they represented a clinical entity to which we gave the name, "syndrome of glaucomatocyclitic crises." Since the original report our experience has broadened, both through personal observations and the reports of other ophthalmologists who have confirmed our findings and helped to strengthen our belief that this is, in fact, a unique form of glaucoma which has a benign prognosis and which calls for a special therapeutic approach, different from that used in any other form of glaucoma.

Kronfeld[4] intuitively sensed these unique characteristics when he referred to "cases of acute elevation of intraocular pressure occurring simultaneously with mild recurrent cyclitis." Even though he did not go into further detail, we believe that he may have had reference to a similar group of cases.

Before describing the clinical characteristics of the syndrome in detail, we wish to point out that it takes an intermediate position between primary and secondary glaucoma. By primary, we mean the acute or recurrent narrow angle glaucoma in which an iris block mechanism is presumed to be the initiating cause. Secondary glaucoma, as used here, refers to the glaucoma associated with relatively severe uveitis in which there is an obvious inflammatory or mechanical basis for the obstruction to aqueous outflow.

The syndrome of glaucomatocyclitic crises, while it may resemble an attack of primary narrow angle glaucoma — especially when cells and keratic precipitates are either absent or masked by the presence of corneal edema — differs from acute glaucoma by occurring in the presence of a normal and open angle. In fact, Chandler[2] stresses this anatomic finding as an important distinguishing characteristic between acute narrow angle glaucoma and the syndrome of glaucomatocyclitic crises. This would be of only theoretical importance were it not for the fact that the treatment of the one disease may be diametrically opposed to that of the other.

In differentiating this syndrome from the commonly encountered forms of glaucoma secondary to uveitis, there are two chief considerations: (1) In the syndrome of glaucomatocyclitic crises, the glaucoma is not a result or complication of the uveitis, but both manifestations are concomitant expressions of a

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single disease entity. Since the individual attacks are self-limited and are separated by remissions during which the eye is apparently normal, it is illogical to implicate foci of infection as a cause. Also, an attack results in no structural changes which could be held responsible for the occurrence of a subsequent attack. (2) From the point of view of therapy, the differentiation is even more vital. In the presence of a high tension and an acute uveitis there is a tendency to resort to surgical intervention, even if only to perform a paracentesis. The value of recognizing this syndrome lies in the fact that surgical procedures are not only unnecessary but definitely contraindicated in this condition.

The clinical characteristics of this syndrome were described in detail in our first paper. More recently, Theodore[7] published an excellent analysis of this syndrome based on personal experience with eight cases. He confirmed our original observations in all essential details. He stresses the importance of a careful slit lamp study in every case of glaucoma because a number of his cases had been mistaken for primary glaucoma. He suggests allergy as a possible etiologic factor.

The homogeneous nature of this disease is borne out by the fact that it has not been necessary to change any of the essential diagnostic criteria since our first report, despite the large number of cases seen since that time by many ophthalmologists in various parts of the country. It should, therefore, suffice to summarize briefly the essential features which characterize the syndrome of glaucomatocyclitic crises.

CLINICAL CHARACTERISTICS

1. The disease is unilateral and the same eye is always affected.

2. During an attack, the affected eye has the wider pupil. Between attacks, the anisocoria may persist.

3. The commonest presenting symptom is slight discomfort, but as a rule the patient does not complain of pain, even at the height of the attack. Blurring of vision and colored halos, when present, indicate a relatively high tension (usually 45 mm. Schiötz, or over). Some episodes may occur without symptoms. The visual acuity is normal, unless it is affected by the presence of corneal edema. In the average case, it does not drop below 20/40.

4. The eye is white, although occasionally mild conjunctival injection is present. Scleral congestion is noted only in the presence of high tension. Those cases which had pain and congestion when first seen had been previously treated with either strong miotics or strong mydriatics, or both.

5. Ocular hypertension usually appears simultaneously with cells in the aqueous. However, the increase in pressure may either precede or follow the onset of cyclitis by one to three days. Exceptionally, individual attacks have been noted, in which the glaucoma was not associated with evidence of uveitis. Similarly, episodes may occur in which cells and even keratic precipitates are found without an accompanying elevation of tension.

6. Individual attacks of ocular hypertension may last from a few hours to one month, but very rarely over two weeks. The tension fluctuates. It may rise as high as 90 mm. Schiötz, but the most frequently observed initial tensions are between 40 and 60 mm. Hg because tensions lower than 40 mm. Hg are generally unaccompanied by symptoms. The end of an attack is often heralded by a drop of tension to a level below that of the other eye.

7. Keratic precipitates are generally noted within three days of the onset of the attack. If they are small, they may be obscured by the presence of corneal

edema, in which case the instillation of a drop of glycerin preceded by pontocaine will aid in their detection. The keratic precipitates are small, discrete, and not pigmented. They are flat, round, and not of the type commonly referred to as "mutton fat." They are few in number, seldom over twenty-five, and are most numerous in the lower third of the cornea. They begin to disappear soon after they are formed. Fresh ones may appear, however, with each exacerbation of the hypertension. After the subsidence of the hypertension, the precipitates fade away, within a few days to a month. At no time does one see more than a trace of an aqueous flare. Posterior synechias are never formed.

8. The anterior chamber in these cases exhibits the same variations as in normal eyes. We have not encountered any case with an excessively narrow angle in which the latter could be regarded as the cause of the glaucoma. Even at the height of an attack, each of the cases studied gonioscopically appeared to have an open angle.

9. Heterochromia was noted in 3 of the 9 cases reported in the original paper, but has not been observed in any of the subsequent cases. It, therefore, cannot be regarded as a characteristic feature of this syndrome. When present, it may be an expression of involvement of the sympathetic nervous system.

10. Episodes may occur with varying frequency and without any apparent cause. Between attacks, the eye is completely normal and gives negative responses to all types of provocative tests. We can corroborate Grant's observation[3] that during the attack the facility of outflow as represented by Grant's coefficient is abnormally low.

11. The visual fields show no permanent changes attributable to the recurrent attacks of ocular hypertension. Transitory enlargement of the blind spot through a widening of the angioscotoma tree may be noted when the tension is high.

TREATMENT

Treatment of the ocular hypertension should be limited to the attack and should consist in the use of mild miotics, such as pilocarpine ½ to 2 per cent.[6] Topical cortisone has a beneficial effect on the course of the attack. The value of cortisone was first pointed out by Theodore[8] in 1950. Frequent dilatation with phenylephrine 10 per cent, carried out in the office, helps to reassure the physician that no synechias are forming and may also effect a temporary lowering of the tension. No treatment is necessary between attacks, since miotics do not prevent recurrences. When feasible, it is of advantage to observe the course which the attack takes without the use of active therapy, since each episode is self-limited. Strong miotics, and perhaps also strong mydriatics, tend to aggravate the symptoms by producing pain, congestion or spasm of the ciliary muscle.

Various surgical procedures have been tried, namely paracentesis, basal iridectomy, peripheral iridectomy, iridencleisis and trephine. All of these operations have been unsuccessful in preventing recurrences. Billet[1] reported a case which had been treated as primary glaucoma by two ophthalmologists, one performing a trephine and the other an iridencleisis. Though the operations were surgically well executed, the attacks continued to recur and showed the same characteristics as before surgery. Theodore[7] also mentions two cases where surgery was performed by other ophthalmologists, without influencing the subsequent course of the disease. Similar experiences have been reported to us by several other ophthal-

TABLE I

DIFFERENTIAL DIAGNOSIS OF THE SYNDROME OF GLAUCOMATOCYCLITIC CRISES

ACUTE NARROW ANGLE GLAUCOMA	GLAUCOMATOCYCLITIC CRISES	GLAUCOMA SECONDARY TO UVEITIS
Usually bilateral	Unilateral; recurrences always in same eye	Unilateral or bilateral
Pupil wide, irregular and immobile	Pupil slightly dilated, regular and reacts	Pupil small and irregular
Severe pain in congestive stage	No pain or slight discomfort	Variable degree of pain
Vision markedly diminished in congestive phase	Vision normal or slight blurring in presence of corneal edema	Impairment of vision depends chiefly on degree of uveitis
Various degrees of scleral congestion	Eye is white	Various degrees of ciliary injection
Usually no keratic precipitates; pigment dust may be present	Cells in anterior chamber and up to 25 unpigmented keratic precipitates	Cells in anterior chamber; keratic precipitates more numerous
Aqueous ray may be present	Aqueous ray absent or minimal	Marked aqueous ray
No posterior synechias	No posterior synechias	Usually posterior synechias
Narrow angle; closed during attack	Angle shows normal variations in width and is open during attack	Angle open but may contain particulate matter
Dark room test usually positive	Dark room test negative	Dark room test negative
Pupil may be enlarged and irregular. Iris atrophy may be present	Pupil frequently slightly larger; but otherwise normal. No posterior synechias or iris atrophy	Pupil irregular, posterior synechias frequently present; may show iris atrophy
Pigmented deposits on cornea and lens may persist	Keratic precipitates persist for variable time up to one month after tension is normal	Pigmented and nonpigmented keratic precipitates may remain as long as uveitis is active
Iridectomy prevents recurrences	Iridectomy and filtering operations do not prevent recurrences	Surgery may help in some cases

Note: Acute narrow angle glaucoma in the noncongestive phase may resemble glaucomatocyclitic crises. The important differential points are (a) an extremely narrow anterior chamber angle as compared with a normal and open anterior chamber angle in glaucomatocyclitic crises, and (b) the absence of keratic precipitates in the noncongestive phase of acute narrow angle glaucoma.

mologists. In view of the ineffectiveness of surgical measures and the benign and self-limited nature of the disease, there exists no indication for any surgical intervention in this syndrome.

DIFFERENTIAL DIAGNOSIS

While this syndrome represents a group having a uniform symptomatology, there undoubtedly are cases which are commonly diagnosed as primary glaucoma or as secondary glaucoma and which are in some respects related to glaucomatocyclitic crises. Our limited experience, however, does not enable us to analyze these differences more fully at this time. For example, we have seen at least three patients with marked iridocyclitis displaying ciliary injection, posterior synechias, and numerous keratic precipitates associated with a persistent elevation of tension in whom the symptoms subsided in four to six weeks without surgical intervention and with complete restoration of ocular function. Even more frequent are cases of acute congestive and noncongestive glaucoma which satisfy most of the criteria for primary glaucoma except that the angle of the anterior chamber is open. Such atypical cases are difficult to classify, and prolonged observation may be required in order to determine the best approach to therapy. As a general rule, it is advisable to proceed cautiously, employing conservative treatment and using the indicated miotics or mydriatics in low concentrations.

Differential diagnosis of the syndrome of glaucomatocyclitic crises from both secondary and primary glaucoma may be facilitated by arranging the symptoms and signs in parallel columns (table 1). While a given case may not satisfy all of the criteria set down in this table, the general picture created by the symptomatology will delimit these three conditions from one another.

SUMMARY

1. The syndrome of glaucomatocyclitic crises is presented as a clinical entity which should be differentiated from other forms of glaucoma.

2. It consists of recurrent attacks of unilateral acute glaucoma associated with signs of mild cyclitis.

3. During an attack, the eye is white and pain is minimal despite marked elevation of tension. A few keratic precipitates are present and the pupil of the affected eye is larger than the other.

4. The attack is self-limited. Visual acuity and field remain unimpaired.

5. The angle of the anterior chamber is open, even at the height of the attack. This differentiates it from narrow angle glaucoma.

6. Between attacks, all provocative tests give normal responses and facility of outflow is normal.

7. Treatment should be limited to mild miotics and topical cortisone. No treatment is necessary in the interval between attacks.

8. Surgical intervention does not prevent recurrences.

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DISCUSSION

PETER C. KRONFELD, M.D., Chicago, Ill.: Although this has been said a good many times before, I would like to say again that Dr. Posner and associates were the first ones officially to recognize and record this disease as an entity. By doing so they contributed very materially to the clarification of an important issue. The term "glaucomatocyclitic crises" seems to have been well chosen and has established itself in ophthalmologic terminology. The paper to which we had the pleasure of listening today is a concise presentation of present clinical knowledge concerning this disease. We have a number of such cases under observation and in most of them the disease is running true to form, the relationship between cyclitis and glaucoma being just as described by Dr. Posner. I know of no case in which the uveitis in the affected eye got out of bounds, that is assumed a greater magnitude and severity than in the typical cases. This cyclitis, according to present clinical standards, is real and not just a phenomenon of repair or recovery from an acute vascular crisis. In this respect, as in so many other respects, the syndrome of glaucomatocyclitic crises differs from acute narrow angle glaucoma, which often leaves in its wake an iritis-like picture due to tissue damage and recovery.

We have in our clinic one case in which after many, apparently typical attacks, the optic nerve showed signs of damage, which has continued at a very slow rate while the patient has been under our observation. This patient, however, has been atypical in the sense that he has had a fairly severe hypertensive cardiovascular disease which was first noted at the age of 30. The fundus picture has been that of a primary optic atrophy with atrophic excavation. The entire arterial tree is somewhat narrow in both eyes. The visual

field defects have been of the usual glaucomatous pattern. The optic nerve findings in this case of glaucomatocyclitic crises are in striking contrast to those in all others, which have weathered innumerable attacks without the slightest optic nerve damage.

Although a good many of our patients have been examined from head to toe, we have no clues to the etiology of this disease nor do we know of any measure that will prevent the attacks.

I have been interested in the mechanism of the elevation of tension in these cases but have not reached conclusive results. Between the attacks, these eyes seem to have a normally regulated ocular tension, all ordinary provocative tests giving a normal result. During the attack a serious outflow disturbance appears to be in the foreground. The gonioscopic picture remains normal, so the trabeculum in all probability functions normally. The aqueous veins appear to carry blood at the time of the attack.

Drs. Posner and Schlossman in today's paper have stressed the differential diagnosis which is probably the most important practical aspect of this disease. It is the ophthalmologist's prime duty and function to distinguish it from acute narrow angle glaucoma and from the various chronic glaucomas secondary to uveitis. The tabulation of the characteristic symptoms of each of those three conditions I am sure will prove very helpful to all of us. Let us hope that the continued study of these cases will reveal more definite information concerning the pathogenesis, etiology and treatment of this disease.

HAROLD G. SCHEIE, M.D., Philadelphia, Pa.: There is no doubt as to the validity of the group of findings known as glaucomatocyclitic crises, described by Posner and Schlossman in 1948 and reviewed by them again today. The condition is especially important because it is frequently overlooked, often to the patient's great disadvantage.

I should like to discuss one or two points and to raise one question. First, the authors have helped resolve one of the important arguments between the gonioscopic and the neurovascular approaches to glaucoma. Adherents to the gonioscopic school believe that acute, congestive glaucoma occurs only when the angle of the anterior chamber closes due to iris block. However, occasional confusing eyes are seen, where apparently acute glaucoma occurs in eyes with open angles. The number has been estimated at anywhere from 2 to 20 per cent; I believe W. Stewart Duke-Elder now believes it to be more nearly 2 per cent. If this small group of cases, which seems to be responsible for a breakdown in the gonioscopic concepts of acute glaucoma, falls into the syndrome described by Posner and Schlossman, as I believe they do, I feel the authors have made a very real contribution.

The other point I would like to emphasize is the favorable prognosis mentioned by the authors and by Dr. Kronfeld. In general, these patients do very well regardless of the type of treatment. I have given some mydriatics and all seem to recover. Avoiding surgery is of the greatest importance and, in general, that is true of all types of glaucoma secondary to uveitis. For some reason eyes with secondary glaucoma of this type tolerate increased tension much better than do eyes with primary glaucoma.

Finally, I would like to raise the question about whether or not this group of findings should be set aside as a truly separate disease entity. Although the findings are characteristic, these patients all show signs of a mild anterior uveitis. I would prefer to believe that this group of findings represented an unusual response to a mild uveitis or iridocyclitis where for some reason the tension rises very rapidly with a minimum of inflammatory signs.

PATHOLOGIC FINDINGS IN AN EYE WITH A NONHEALING CATARACT INCISION

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THIS report concerns the eye of a woman aged 69. A successful cataract extraction had been performed on one eye. Approximately one year later a cataract extraction was done on the other eye. There were no operative complications but the second eye failed to heal and this report concerns the nonhealing eye.

On April 20, 1950, the cataract extraction was done on the left eye. This was an intracapsular extraction with a peripheral iridectomy and the eventual visual acuity was 6/6+. There were no complications either at operation or postoperatively. The patient had an easy convalescence and learned to use her cataract glass without difficulty.

On July 6, 1951, an intracapsular cataract extraction was done on the right eye. There was a peripheral iridectomy; there was no vitreous loss. The incision was closed with two mild chromic catgut sutures. The edges were approximated evenly and without tension.

On July 14, 1951, the patient experienced sharp pain in the operated eye. Examination indicated slight incarceration of the iris at the temporal pillar. The temporal suture was loose but there was no hemorrhage in the anterior chamber. The iris incarceration enlarged and an iris prolapse developed. On July 16, 1951, the iris prolapse was excised and the incision was closed with two silk corneal-scleral sutures. This was done under pentothal anesthesia so that careful surgery could be carried out.

Again the prolapse occurred and the incision was repaired on July 31, 1951, again on August 9, and again on August 13, 1951. Silk sutures were used at all times. Plasma and thrombin were also used. The incision failed to heal, and because of some pain which developed eventually, enucleation was done on August 27, 1951.

The patient was a diabetic. The diabetes was mild and easily controlled and she was on an adequate diet. The total protein measured 7.2, albumin was 5.2 and globulin 3.0. This gave an albumin-globulin ratio of 1:1.7. The coagulation time was two and one half minutes; the bleeding time, one minute. Urinalysis on repeated examinations revealed nothing of significance. The blood sugar varied from a low of 82 to a high of 126 milligrams per 100 cc. The blood cholesterol was 200 mg. per 100 cc. and the Wassermann and Kahn tests were negative. The blood count showed 75 per cent hemoglobin, 4,100,000 red blood cells. The patient was at one time given a transfusion of 500 cc. of blood. There was no history of sensitivity except to phenobarbital. The eye was sectioned vertically so as to include the incision.

At one side of the cornea the incision could be seen. There was a space between the two cut ends of the cornea

From the Wills Eye Hospital, Philadelphia Pa.
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and in this space were a number of proliferating epithelial cells with a few fibroblasts, but the ends showed no evidence of healing. In the remainder of the cornea, the epithelium was intact. There was a small amount of exudate between the epithelium and Bowman's membrane. Bowman's membrane was intact.

The stroma showed a minimal degree of vascularization at the edge away from the incision with somewhat more vascularization on the side of the incision and some subconjunctival hemorrhage. The stroma in general did not show any increase in the stroma cells. There appeared to be, if anything, a diminution in the number of stroma cells in the area near the incision. Descemet's membrane and the endothelium were present.

The anterior chamber had collapsed. At places a minimal degree of exudate could be seen in the anterior chamber and at one side there were some proliferating epithelial cells. The iris structure was condensed but did not show any areas of infiltration.

There was moderate hyperemia with no cellular infiltration.

The suprachoroidal space was filled with hemorrhage and the underlying choroidal structure showed separation by a hemorrhagic infiltration. The retina was normal.

A diagnosis of subchoroidal hemorrhage was made.

SUMMARY

The history and important pathologic findings of a case in which a cataract incision failed to heal have been presented. The patient exhibited no unusual constitutional changes and there were no complications due to technic at the time of operation or subsequently at the time of repair. The patient was cooperative and there was no history of trauma adequate to explain the nonhealing. The only pathologic finding of importance was the presence of hemorrhage in the subchoroidal space which was not massive in extent. The apparent cause for this nonhealing was a subacute subchoroidal hemorrhage.

SELECTED PROBLEMS IN THE DIAGNOSIS OF LARYNGEAL CARCINOMA

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THIS report is the direct outgrowth of efforts to photograph various types of laryngeal pathology for teaching purposes. In the course of accumulating and reviewing pictures we were impressed by the problem of leukoplakia in its varied forms and the associated incidence of vocal cord carcinoma. The relative lack of symptoms in extracordal or extrinsic cancer in view of the amazing extent to which such lesions had often progressed before recognition was a consistently disturbing observation. We became interested in correlating mirror and direct laryngeal views with operative specimens of the same patient. The striking similarity between the gross appearance of some forms of laryngeal tuberculosis and carcinoma was evident on numerous occasions. Inasmuch as the choice of therapy often involved problems in diagnosis, some consideration of treatment has been included. For a more definitive analysis of these particular topics than was afforded by photography, we have gathered from our records such statistics and clinical data as may seem pertinent.

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MATERIAL AND CLASSIFICATION

The study is based upon a series of 116 patients with carcinoma of the larynx seen in the past five years.

We have followed, with minimal modification, the classification proposed by Walsh.[29] To avoid varied interpretations of the term "intrinsic carcinoma," however, we prefer the designation "cordal carcinoma" for tumors confined to either one or both true vocal cords, with normal cord mobility.

When a tumor involves the true cord and the ventricle or the ventricular band, or extends just inferiorly from an initial cordal origin, we favor the term "endolaryngeal" to distinguish it from cordal carcinoma. In most endolaryngeal carcinomas the true cord is fixed, or its motion impaired, although this is not uniformly true.

A distinct and separate entity is the type of growth which arises primarily in the subglottic space. Such tumors may extend superiorly to involve the true cord or inferiorly to invade the upper trachea.

Finally, the terms extracordal or extrinsic are used synonymously to denote a primary lesion of the epiglottis, arytenoid, aryepiglottic fold or pyriform sinus. This group comprises the largest category of laryngeal malignancies in our series, and poses the greatest difficulty with respect to early recognition and adequate therapy.

Laryngeal neoplasms are sometimes so extensive that their site of origin can-

TABLE I

ANALYSIS OF LARYNGEAL CARCINOMA (116 PATIENTS)

CLASSIFICATION	NUMBER	PER CENT
Cordal carcinoma (limited to true cords, normal mobility)	31	26.7
Endolaryngeal carcinoma (true cord, ventricle, false cord or early subglottic invasion)	18	15.5
Subglottic carcinoma	9	7.7
Extracordal (extrinsic) carcinoma (epiglottis, arytenoid, aryepiglottic fold, pyriform sinus)	58	50.0

not be ascertained. Under such circumstances, a careful history may aid in determining the original location.

In the entire series there were 113 male patients and 3 females. (The incidence of carcinoma of the larynx in females varies from 3 to 10 per cent. Our series includes a number of males seen in a Veterans' Hospital.) The average age for the group was 56.7 years, although for patients with subglottic carcinoma this figure was somewhat higher.

It will be observed from table I that the ratio between intralaryngeal and extrinsic lesions corresponds to the range of distribution reported by other authors.[16,17]

CORDAL CARCINOMA

As previously stated, this category embraces only those lesions which are limited to the true cord (figs. 4, 5, 6, 13), may or may not traverse the anterior commissure and do not interfere with normal mobility. There were 31 patients in this group, including the only three females represented in the entire series. The average age of the group was 56.2 years. Hoarseness was the only symptom and was present in every patient.

Each member of the group except one gave a history of smoking, and two thirds of these could be regarded as heavy smokers (in excess of one package of cigarettes daily). The incidence of smokers is thus considerably higher than the 73.7 per cent figure quoted by Wynder and Graham[31] as representing the smokers among a general (non-cancer) hospital population.

The duration of the symptom of hoarseness before diagnosis averaged seven months. (This figure does not include 20 per cent of the group who had been hoarse for years.) Six of the 31 patients had been treated with antibiotics, sprays, gargles, lozenges and the like during the period when it could be presumed that evidence of malignancy might well have been at hand. The factors of patient neglect and shortcomings of the medical profession, as outlined by Pack and Gallo[23] and Robbins and co-workers[25] are apparent in these statistics. Ferguson[5] has recently suggested that the widespread use of antibiotics may be responsible for a definite trend away from early diagnosis in laryngeal cancer. The delay in diagnosis in this series emphasizes Ferguson's contention. One of the patients in this group showed a second primary malignancy. None had palpable cervical lymph nodes.

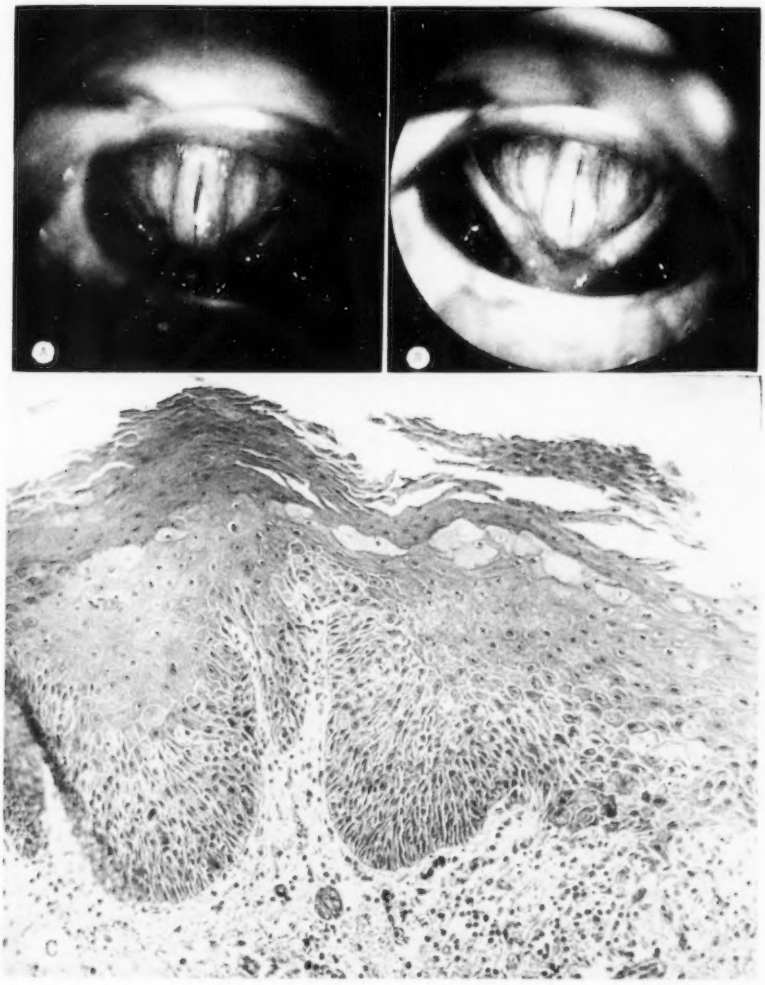

FIG. 13—A, Leukoplakia involving middle third of left true cord. Biopsy: epithelial hyperplasia. B, Recurrence of leukoplakia in same patient anterior to original site. C, Same patient. Section through vocal cord removed by thyrotomy: hyperkeratinization, epithelial hyperplasia and carcinoma in situ (hematoxylin and eosin, x90).

Leukoplakia and Cordal Carcinoma
(figs. 3, 4, 5, 13, 14)

Among the 31 patients with cordal carcinoma there were 19 (61 per cent) who had definite evidence of leukoplakia, and in 9 of the 19 the condition was bilateral. The majority of these individuals were excessive smokers (two packages of cigarettes or more per day). LeJeune[15] has noted that leukoplakia is being seen more frequently than in former years and that all of his patients with leukoplakia of the vocal cords were

excessive users of tobacco. Similar observations on the relationship between leukoplakia and excessive tobacco use have been made by Clerf, Putney, and O'Keefe.[3]

Unless marked infiltration of the vocal cord has occurred, it is often difficult to evaluate the status of a leukoplakic site. This was ably described by Negus[18] in 1939: "Localized or diffuse overproduction of the superficial epithelium offers considerable difficulty in deciding whether malignant changes are commencing in the deeper layers of what is a potentially precancerous condition." As an illustration, the following case may be cited:

T. W., a 59-year-old male, had been hoarse for five months. There was leukoplakia of the free edge of the middle third of the left vocal cord (fig. 13A). Three biopsies were reported as showing only epithelial hyperplasia. Because of the recurrence of leukoplakia anterior to the original site (fig. 13B), thyrotomy was performed and the vocal cord was removed. Serial sections disclosed epithelial hyperplasia along with carcinoma in situ but no subepithelial invasion (fig. 13C). The patient died an accidental death two years later without clinical evidence of recurrence.

It is our feeling that an active plan of treatment should be undertaken where any reasonable doubt as to the possibility of cancer exists.

The role of chronic irritation in precancerous conditions of the larynx has been emphasized repeatedly by Jackson and Jackson.[13] Clerf refers to keratosis, hyperkeratosis or leukoplakia as primarily a localized benign hyperplasia of the epithelium which does not invade the submucosa.[2] In discussing etiology he points to the predominance of the condition in males and to the excessive use of tobacco (although there is no definite proof that this is a causative factor). Alcohol is of doubtful influence. Most of the patients use their voices excessively or abnormally. Chronic laryngitis is a well-recognized predisposing factor, and this has been substantiated by New and Erich's histologic demonstration of the marked subepithelial inflammatory reaction seen in these conditions.[19] Dietary deficiency has been thought to be of significance in some individuals.[8]

While leukoplakia and keratosis are usually considered in discussions of benign growths in the larynx,[19] it is equally true that references to them are found in almost all dissertations on laryngeal malignancy.[11] Their intimate relation to cancer and the necessity for frequent examination of the larynx in these patients cannot be overemphasized.[7] It is well known that simple keratoses may be removed and not recur, that they may reappear in benign form after removal in some patients, and that the changes of malignancy may supervene slowly or rapidly in others. There is no way of predicting the sequence of events in any given situation.

W. C., a 54-year-old male and an excessive smoker, had been hoarse for nine months. During most of this period he had receive antibiotics for his voice difficulty. A whitish proliferative area was noted on the anterior half of the left true vocal cord (fig. 1). This was removed and showed epithelial hyperplasia, hyperkeratinization, and a submucosal inflammatory reaction. No recurrence has been observed in over three years.

The rapid development of malignancy upon a leukoplakic anlage is illustrated by the following case:

L. Z., a 55-year-old male with a history of heavy smoking for years, had been hoarse for only three months. On laryngeal examination there was observed a white granular elevation along the superior surface of a markedly thickened right true vocal cord (fig. 5). Motion of the cord was not impaired. There were also leukoplakic alterations on the opposite cord. Biopsy of the right cord disclosed squamous cell carcinoma arising in an extensive leukoplakic infiltration.

In contrast, malignant transformation may occur after a long interval.

C. H., a 44-year-old male salesman and heavy smoker, gave a history of having been hoarse for eight years. He had undergone a stripping operation on his right vocal cord six years previously and had been informed that the tissue had shown leukoplakia. When seen in 1949 there was a whitish mass involving the right true cord, extending posteriorly to the vocal process of the arytenoid and anteriorly to the commissure, where it projected slightly (fig. 4). Some subglottic thickening was evident but mobility of the cord was not impaired. Biopsy showed squamous cell carcinoma. The entire larynx was reddened and there was leukoplakia of the opposite cord. There were no palpable cervical nodes. Laryngectomy was performed in 1949. It was found that the lesion extended just across the anterior commissure, invaded the floor of the ventricle and reached the arytenoid posteriorly. The patient has remained well since surgery.

Four patients with similar histories were carefully followed by Clerf for two to four years, of which 3 eventually required partial laryngectomy and 1 a complete laryngectomy.[2]

Pachydermia of the larynx represents another form of diffuse epithelial hyperplasia with potential malignant alteration. This condition usually appears in the posterior commissure but may later become evident along the entire glottic rim.[9]

C. S., a 58-year-old male, had been intermittently hoarse for one year and persistently so for six months prior to admission. There was a whitish, pebbly irregularity involving both true vocal cords and the interarytenoid space, with normal cord mobility (fig. 14A). A diagnosis of carcinoma was made on the fourth biopsy; the previous ones showed only epithelial hyperplasia. The laryngectomy specimen revealed diffuse carcinomatosis limited to the true vocal cords and interarytenoid space (fig. 14B). The patient has remained well two and a half years after surgery.

The prognosis of cancer of the true vocal cord is excellent, probably because of the early appearance of voice change and the relatively scant lymphatic drainage.

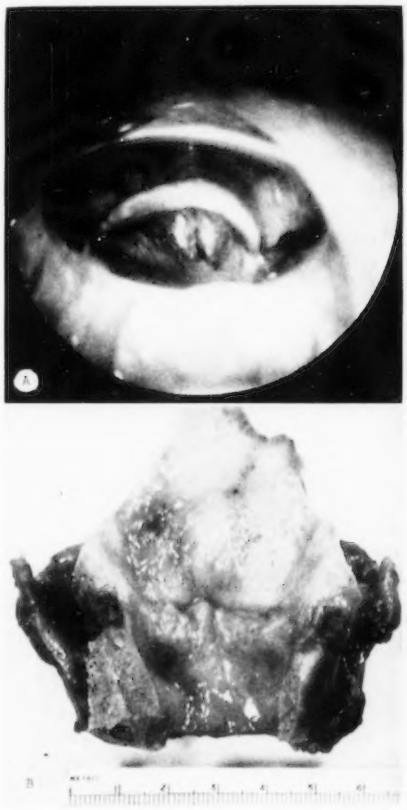

FIG. 14.—A. Pachydermia of the larynx with whitish pebbly irregularity of both vocal cords and normal mobility. Fourth biopsy revealed carcinoma. B. Laryngectomy specimen of same patient. Bilateral carcinomatosis of true cords and interarytenoid space.

Endolaryngeal removal of certain small localized lesions, irradiation, and partial or total laryngectomy all have their proponents and specific indications, but the careful selection of cases is the principal assurance of success in dealing with cordal lesions. The already high rate of cure of cordal cancer will probably be improved if, through education, we can shorten the average seven-month interval between the onset of hoarseness and the inception of defini-

tive care, if adequate examination is substituted for inapplicable therapeutic measures, and if laryngologists insist on the guarded scrutiny of patients with leukoplakia, keratosis and pachydermia, the so-called precancerous conditions involving the larynx.

ENDOLARYNGEAL CARCINOMA

This group is represented by tumors which involve the true vocal cord and the ventricle, the ventricular band, or the immediate infraglottic area (figs. 4, 15, 16). Impaired mobility or fixation of the true cord is often present, though not invariably so. There were 18 such patients with an average age of 55.2 years. All were males, the symptom of hoarseness was present in each but, in contrast to cordal carcinoma, 6 patients or 33 per cent, also mentioned the sensation of throat discomfort. Smoking was recorded in the history of all but 4, upon whom this information was not available. The average duration of symptoms before diagnosis was 8.7 months. Only 2 patients had been treated irrelevantly before admission. Leukoplakic alterations were evident in the larynges of 4, all heavy smokers. One patient had pulmonary tuberculosis in addition to carcinoma of the larynx. None of the patients had palpable cervical nodes nor were there any in whom a second primary malignancy was evident.

Whereas it is not difficult to judge fairly accurately the extent of a cordal carcinoma, the degree of involvement of an endolaryngeal tumor is more complex and becomes a matter of extreme importance with respect to therapy. Thus we endeavored to correlate the laryngeal appearance with photographs of operative specimens in an effort to establish some familiarity with this type of evaluation.

H. F., a 70-year-old male who had been an excessive smoker for 50 years, had noticed hoarseness for six weeks. Mirror, direct and suspension laryngoscopy disclosed a proliferative lesion of the right true vocal cord throughout its entire length, with impaired mobility but no palpable cervical nodes (fig. 15A). The operative specimen after laryngectomy revealed extension to the ventricle, false cord, base of the epiglottis and across the anterior commissure (fig. 15B). It was evident that the tumor was more extensive than had been suspected clinically.

LEGENDS FOR COLOR PLATE

FIG. 1.—Keratosis of the larynx involving anterior half of left true cord. No recurrence three years after removal.

FIG. 2.—Extensive hyperkeratotic papilloma of left true cord.

FIG. 3.—Larynx of a patient who had smoked excessively for 54 years. Bilateral leukoplakia, with subglottic mass at anterior commissure which yielded positive biopsy for carcinoma.

FIG. 4.—Leukoplakia and infiltration of anterior portion of right true cord in a heavy smoker. (Right cord stripped for leukoplakia six years previously.) Biopsy: carcinoma.

FIG. 5.—Leukoplakia and infiltration of right true cord of an excessive smoker with hoarseness for three months. Biopsy: carcinoma arising in leukoplakia.

FIG. 6.—Cordal carcinoma, with lesion limited to anterior half of left true cord. Normal mobility. No recurrence after thyrotomy in which involved anterior commissure and part of opposite cord were excised.

FIG. 7.—Lesion of the epiglottis of patient with pulmonary tuberculosis, discovered when patient requested removal of ear wax. Biopsy: carcinoma.

FIG. 8.—Proliferative carcinoma on laryngeal surface of epiglottis in patient with diabetes, gangrene and severe cardiac disease.

FIG. 9.—Postirradiation view of previous larynx. All evidence of tumor has disappeared. There has been no recurrence for three years.

FIG. 10.—Extensive carcinoma of left pyriform sinus in a patient with painless cervical node and no other symptoms.

FIG. 11.—Infiltration and ulceration of right arytenoid in an individual with symptoms of two weeks' duration. Biopsy: squamous cell carcinoma.

FIG. 12.—Infiltration and ulceration of left arytenoid and aryepiglottic fold in a patient with pulmonary tuberculosis. Lesion completely disappeared after streptomycin therapy.

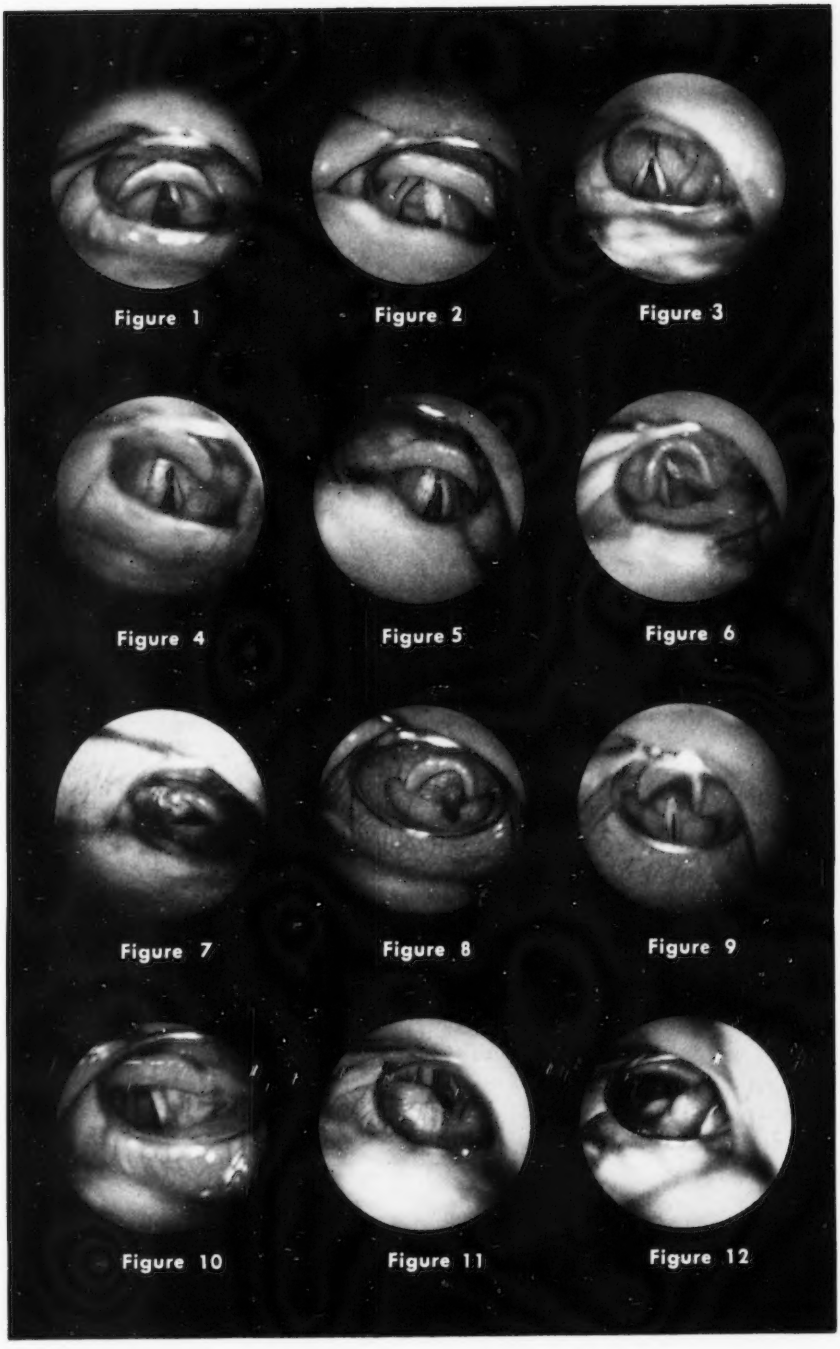
Figure 1
Figure 2
Figure 3
Figure 4
Figure 5
Figure 6
Figure 7
Figure 8
Figure 9
Figure 10
Figure 11
Figure 12

FIG. 15—A. Suspension laryngoscopy view of proliferative lesion involving right vocal cord. Impaired motion of cord was apparent clinically. B. Operative specimen of same patient revealing extension to ventricle, ventricular band, base of epiglottis and across anterior commissure. Endolaryngeal carcinoma.

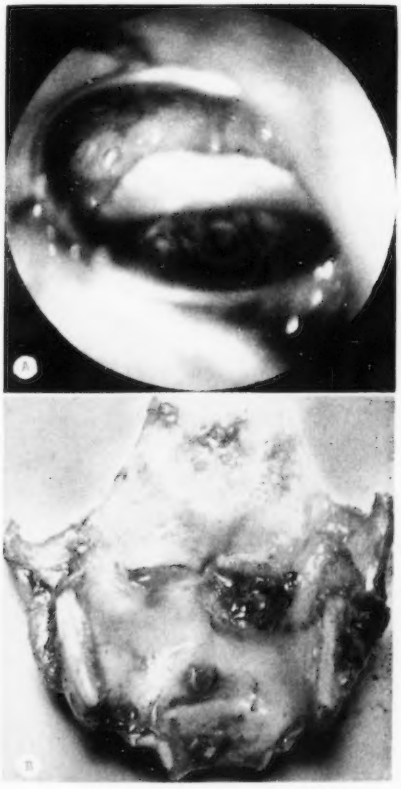

FIG. 16—A. Ulcerating carcinoma of right hemilarynx with subglottic extension. B. Operative specimen on same patient. Good correlation with preoperative estimate of lesion. Endolaryngeal carcinoma.

Another example of this attempted correlation is illustrated by the following:

J. M., a 54-year-old male, had been a heavy smoker for forty years. He had been hoarse for two years. Laryngeal examination showed an ulcerative lesion of the right true vocal cord with subglottic extension. The vocal cord was immobile (fig. 16A). There were no palpable cervical nodes. The laryngectomy specimen disclosed an extension into the ventricle and subglottic space which correlated well with the preoperative clinical estimate (fig. 16B).

It is our feeling that repeated examination of any larynx in this category is often desirable and even necessary to properly assess the degree of pathologic involvement prior to the choice of surgery. As diagnostic aids, suspension laryngoscopy and the use of other types of self-retaininig laryngoscopes provide invaluable means of unhurried study,

In addition to routine roentgenograms, planigrams are particularly useful in outlining subglottic extension in these cases.

These added studies might conceivably result in fewer attempts to extend the indications for laryngofissure in a questionable situation, though we hold that such a procedure is rarely if ever indicated in endolaryngeal carcinoma. Of even greater importance, however, is the decisive choice between wide field laryngectomy and laryngectomy combined with radical neck dissection. Subsequent appearance of cervical metastases not clinically evident at the time of the original surgery seems most prevalent in endolaryngeal carcinoma.[3] Recent studies[20] point to the desirability of routine combined laryngectomy and neck dissection for extensive endolaryngeal cancer, reserving wide field laryngectomy for cordal lesions which have involved the ventricular or subglottic areas to a limited degree. It should also be added that conservative or so-called narrow field laryngectomy for extensive endolaryngeal carcinoma is fraught with considerable risk and is mentioned only to discourage its use in these cases.

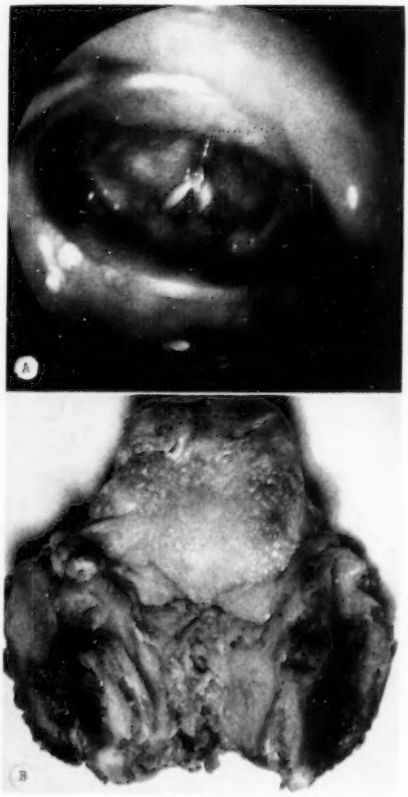

FIG. 17.—A. View obtained by mirror laryngoscopy of subglottic mass obstructing airway, necessitating emergency tracheotomy. Patient had been under treatment for bronchial asthma. B. Operative specimen on same patient, revealing extensive subglottic carcinoma. Additional tracheal rings were removed (not shown on specimen).

SUBGLOTTIC CARCINOMA

Nine male patients comprised this group, the average age being 62.8 years. All had been hoarse and 4 presented evidence of obstructive dyspnea, 3 of these requiring emergency tracheotomy on admission. There was a history of heavy smoking on the part of 6; no information on this point was available on 3 patients. Of particular interest was the 13.4 months' average duration of symptoms before diagnosis. During this interval 2 of the patients had been treated unsuccessfully for bronchial asthma. Leukoplakia was evident in 2 patients. One patient's laryngeal cancer was his second primary malignancy.

Subglottic carcinoma (figs. 3 and 17) does not usually produce symptoms until the lesion has invaded the vocal cord or has caused some narrowing of the airway, as must be evident from the figures in the preceding paragraph. In addition to careful direct laryngoscopy (for these lesions may be easily overlooked on mirror examination), planigrams of the larynx are helpful in outlining subglottic neoplasms.

J. B., a 65-year-old male, had been under treatment for bronchial asthma for nine months. He had been hoarse for seven months. He was known to have diabetes mellitus. Respiratory obstruction and deep cyanosis were evident upon his emergency admission from another hospital. A tracheotomy was performed after rapid visualization of a subglottic mass obstructing the airway (fig. 17A). Wide field laryngectomy was performed eight days later with removal of additional tracheal rings (not shown on specimen) (fig. 17B). Postoperative irradiation was given. Although there has been no evidence of local or metastatic neoplasm in two years, we feel, in retrospect, that laryngectomy and combined neck dissection would have been the procedure of choice in this situation.

Because of the relatively rich lymphatic drainage from the infraglottic area and the frequent delay in diagnosis, subglottic carcinoma is best treated by laryngectomy, or laryngectomy plus block neck dissection. (An indication of the changing concepts in this regard is to be found in the writings of St. Clair Thomson,[27] who reported six cures out of 13 patients with subglottic carcinoma treated by laryngofissure.) Irradiation is regarded as ineffective in subglottic growths:

F. S., a 69-year-old male, had been hoarse for one year. He had been an excessive smoker since the age of 15 and had severe chronic bronchial asthma of an allergic type. There had been one and possibly two episodes of coronary thrombosis. The larynx was characterized by diffuse bilateral cordal leukoplakia and a small subglottic mass beneath the anterior commissure (fig. 3). Irradiation was given because of his extremely poor general condition. There was a resulting chondronecrosis with severe localized pain and dysphagia for a period of eight months before death from inanition. Autopsy disclosed a large area of necrosis involving the larynx and esophagus. Microscopic studies revealed no tumor residue either locally or in the cervical lymphatics.

Although the preceding synopsis is not intended to discredit the merits of roentgen therapy, it is apparent that the dose required for tumor sterilization also resulted in tissue necrosis, with prolonged suffering and eventual death. This might have been avoided by surgery, even though the operative risk was great.

EXTRACORDAL CARCINOMA

In this category are to be found those patients with primary lesions of the epiglottis, arytenoid, aryepiglottic fold or pyriform sinus (figs. 7, 8, 10 and 11). Various terms have been used to designate this group of laryngeal malignancies, among which "extrinsic carcinoma" is probably the most familiar. Orton[21] has suggested that they be referred to as "epilaryngeal tumors." In advocating the expression "extracordal," however, we are thinking of a sharp distinction between these and cordal, endolaryngeal or subglottic growths in order to obviate the varied interpretations hitherto accorded the terms "intrinsic" and "extrinsic." Perhaps an international otolaryngologic organization will some day take up the problem of nomenclature of laryngeal tumors, as was done in the realm of segmental bronchial anatomy.

Of the 58 patients represented in this group, all were males and the average age was 56.3 years. The commonest symptom was some form of throat discomfort which was present in 40 individuals (69 per cent). Among this number 18 complained of actual dysphagia, 7 of localized pain and 2 of a foreign body sensation. Hoarseness was the next most frequent symptom, occurring in 35 patients (60 per cent). There were 47 smokers, 9 indeterminates, and 2 nonsmokers in the group.

In this group of 58 patients with extracordal carcinoma there were 5 who presented two distinct primary malignancies. It will be recalled that 1 patient with cordal cancer and another with subglottic cancer also had second primary lesions. Multiple primary carci-

nomas are by no means rare, and the 6 per cent incidence in our 116 patients is comparable to the figures from autopsy reports on much larger series.[14] Warren and Gates[10] stated, "An individual with one cancer is more apt to develop a second cancer than would be expected by chance alone." This tendency was thought to be eleven times greater than that of a patient without cancer to develop his first lesion.

The average duration of symptoms before diagnosis was eight months. Eight patients (14 per cent) had been treated by measures which indicated inadequate examination.

Considering the abundant lymphatic supply of the extracordal portions of the larynx and the paucity of symptoms due to the relatively silent areas of involvement, it is not surprising to find these lesions far more extensive than either cordal or endolaryngeal growths. Thus, 5 patients in the group had, as their only complaint, painless swelling in the neck (subsequently proven to be metastatic carcinoma). In the entire group of 58 patients, 20 (34.5 per cent) were found to have cervical metastases on admission, and in three instances the metastases were bilateral.

Even more striking in this connection was the discovery of extensive neoplasms in 4 patients undergoing routine hospital examinations for unrelated illness:

A 61-year-old male entered the hospital for repair of a hernia. A firm nodule was noted in both cervical regions. He was found to have a large ulcerating carcinoma of the epiglottis and cervical metastases.

Another patient, also hospitalized for hernia surgery, had unilateral cervical metastasis from a primary carcinoma of the epiglottis.

A third individual, a patient in a tuberculosis sanitarium, requested wax removal from his ear. An ulceration of the epiglottis, presumably tuberculous, was found on biopsy to be squamous cell carcinoma (fig. 7).

The fourth patient with painless cervical swelling was found to have an extensive carcinoma of the pyriform sinus and metastases (fig. 10).

At the present time extracordal carcinoma has the poorest prognosis of any type of laryngeal cancer. There are many patients in whom the results of irradiation are quite remarkable, but it is impossible to predict the response in any given situation. Circumstances sometimes allow no choice.

N. K., a 52-year-old male with amyotrophic lateral sclerosis, was found to have an extensive carcinoma involving the laryngeal surface of the epiglottis and left false cord. He receive irradiation therapy which resulted in complete disappearance of the tumor. Death occurred one year later from his primary illness. At autopsy there was neither gross nor microscopic evidence of any laryngeal tumor or cervical metastasis.

In another instance the patient's general condition was also a contraindication to surgery.

J. A., a 72-year-old diabetic with gangrene and marked arteriosclerotic heart disease, had noted hoarseness and painful swallowing for four months. There was an extensive carcinoma of the buccal mucosa and a secondary primary malignancy involving the epiglottis (fig. 8). Irradiation of the larynx resulted in complete regression of the tumor (fig. 9). The oral lesion was similarly treated. There has been no recurrence of either for two years.

In contrast to these examples may be cited the following:

E. T., a 55-year-old male, noticed throat discomfort and hoarseness for two months. He was found to have an ulcerating carcinoma involving the epiglottis, left aryepiglottic fold and pyriform sinus. Surgery was refused and irradiation was substituted. There was an excellent response for five months, followed by reappearance of tumor on the arytenoid. Combined laryngectomy and block dissection was carried out. Esophageal fistula resulted, which eventually healed, and the patient has been well for two years.

With full realization of the gravity of the prognosis in these cases, current efforts are being made to improve the

cure rate by means of surgery. Certain patients whose lesions were hitherto irradiated as a palliative measure (with hopes but no assurance of a favorable response) are now regarded as likely candidates for en bloc resection of the cancer-bearing areas.

W. B., a 54-year-old male, had noticed hoarseness and throat discomfort for six months. Treatment with gargles and antibiotics had been given ineffectively for four months. On admission he was found to have an extensive carcinomatous ulceration involving the left ventricular band, aryepiglottic fold and pyriform sinus. Laryngectomy and neck dissection were carried out in continuity. The patient has remained free of disease for two years.

More extensive procedures involving resection of the pharynx and esophagus have been recommended by Brunschwig,[1] Orton[22] and others.

In summarizing current thought regarding treatment of extracordal carcinoma, it must be re-emphasized that the prognosis is poor and will probably not improve appreciably until some form of cancer detection test insures earlier recognition. Although there is a definite place for irradiation in cancer of the larynx, the most favorable results reported by radiologists have not compared with surgical figures.[20,21,26] (In all fairness it should be mentioned that surgical figures deal principally with operable cases while radiologic analyses must include operable, inoperable, and recurrent cases.[6]) The cases deemed most suitable for surgery are also those most likely to obtain a good result from irradiation.[4] And finally, the salvage rate with improved surgical management is appreciably greater than figures on the most optimistic irradiation results.

CARCINOMA OF THE LARYNX AND TUBERCULOSIS

Among the 116 patients with carcinoma of the larynx represented in this study are included 6 individuals with pulmonary tuberculosis. Our interest was aroused by the frequent gross similarities between tuberculous and malignant disease of the larynx apparent on color photography. It soon became evident that almost any tuberculous lesion could be matched pictorially by one of proven carcinoma and vice versa.

A 48-year-old male with advanced pulmonary tuberculosis and a normal voice had noticed difficulty in swallowing for one month. Edema and ulceration of the left arytenoid (fig. 12) subsided completely on streptomycin therapy.

A 55-year-old male had been aware of throat discomfort and hoarseness for two weeks. There was a tumor of the right aryepiglottic fold extending to the postcricoid area (fig. 11) with cervical metastasis. Biopsy disclosed squamous cell carcinoma.

Aside from the gross similarity between the two conditions, the diagnostic aspects of carcinoma of the larynx and tuberculosis may be considered under three headings: (1) the coexistence of the two diseases in the same larynx, (2) laryngeal tuberculosis as a possible precursor of cancer, and (3) the differential diagnosis of the two diseases.

Various observers have reported histologic proof of the presence of the two diseases in the same larynx. Jackson and Jackson[12] referred to a number of cases of cancer of the larynx associated with tuberculosis. In the 116 patients with cancer in this series, and in over 200 patients with laryngeal tuberculosis seen by us, the two diseases were never found to coexist in the same larynx. The chief significance of the occasional occurrence of both tuberculosis and cancer of the larynx would seem to be as a medical oddity, though there can be no doubt of their rare simultaneous occurrence.

The possible role of fibrous tissue from tuberculosis or syphilis of the larynx as a precursor of cancer has been

described. There is little clinical evidence to support this relationship. Many of the 200 patients with tuberculous laryngitis have been followed for long intervals after streptomycin therapy, and the effects of healing are being studied histologically. Thus far there has been no instance of carcinoma developing at the site of the laryngeal tuberculosis.

One standard medical school examination question in otolaryngology used to require a differential diagnosis of syphilis, tuberculosis, and cancer of the larynx. In our experience the differential usually rests between tuberculosis and cancer. It is well known that cancer of the larynx may occur at any age, but most commonly between the ages of 45 and 65. One of the most surprising alterations in the age incidence of any disease has been evident in pulmonary tuberculosis. Until recently it was considered a disease of adolescents and young adults. Today the third, fourth and fifth decades are the years of greatest incidence, which approximates the commonest age for cancer of the larynx.

Hoarseness is the most frequent symptom in both cancer and tuberculosis of the larynx. Pain is more likely to be present in tuberculosis than in carcinoma, but exceptions are so common that this is not an accurate differential point.

The gross pathologic alterations of infiltration, ulceration and tumor are common to both diseases (figs. 11 and 12). It might be expected that tuberculosis would be diffuse and bilateral, while cancer would be localized and unilateral. We have found an exact reversal of this situation so frequently that this has not been a reliable differential point.

One important objective finding concerns mobility of the vocal cord. Fixation occurs relatively early with moderate involvement in carcinoma. Extensive infiltration and tumefaction of the arytenoids may be present in laryngeal tuberculosis without impairment of motion of the cords.

The advent of antimicrobial agents effective in tuberculosis offers the possibility of a therapeutic test to aid in the diagnosis. A number of reports[10,24,28] disclose that cancer was suspected only after the lesion failed to improve with the use of streptomycin or para-aminosalicylic acid (PAS). This trial by antibiotic therapy is not recommended because of the valuable time which may be lost if a malignancy exists.

The introduction of streptomycin and other agents has also been important in the diagnosis, since there is now no hesitancy in performing a biopsy in laryngeal tuberculosis. Formerly this might have been deferred for fear of ulceration and the possible deleterious effect on the laryngeal disease. The rapid healing effect of streptomycin in laryngeal tuberculosis removes any possible harmful effects of biopsy.

The presumptive or clinical diagnosis of laryngeal tuberculosis may be established with a high degree of accuracy by the demonstration of the pulmonary disease and the appearance of the larynx. Similarly, ruling out the disease in the chest would eliminate a tuberculous laryngeal lesion. However, the possibilities of error are sufficiently great to warrant biopsy in all laryngeal lesions suspected of being either tuberculosis or cancer.

SUMMARY AND CONCLUSIONS

Some of the diagnostic problems encountered in a study of 116 patients with laryngeal carcinoma have been presented.

The terms "cordal," "endolaryngeal," "subglottic" and "extracordal" have

been used to designate the four principal types of laryngeal carcinoma.

Hoarseness was the only symptom of cordal carcinoma and was present in each of the 31 patients in this category. The duration of this symptom before diagnosis averaged seven months. The widespread use of antibiotics may be a responsible factor in delayed diagnosis of early laryngeal cancer.

Leukoplakia was evident in 19 of 31 patients (61 per cent) with cordal carcinoma. Inasmuch as leukoplakia figures prominently as a precursor of cordal carcinoma and is seen most often in heavy smokers, excessive smoking may be regarded as a predisposing factor.

Hoarseness was uniformly present in 18 patients with endolaryngeal carcinoma. Throat discomfort of varying degree was also noted by 6 patients in this group.

The diagnosis of subglottic carcinoma was often obscure until invasion of the vocal cord or narrowing of the airway occurred. The average duration of symptoms before diagnosis in subglottic carcinoma was 13.4 months, an interval appreciably greater than in other forms of laryngeal carcinoma.

Extracordal carcinoma developed silently or with minimal symptoms. The commonest symptom was some form of throat discomfort, present in 69 per cent of the patients.

In the entire group of 58 patients with extracordal carcinoma, 20 (34.5 per cent) were found to have cervical metastases on admission.

Multiple primary malignancies were found in 7 of the 116 patients in this study.

Tuberculosis of the larynx and carcinoma of the larynx often presented comparable gross appearances. Biopsy in laryngeal tuberculosis is warranted when cancer is suspected.

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DISCUSSION

HENRY B. ORTON, M.D., Newark, N. J.: First I wish to congratulate the authors on their presentation and beautiful photography. I cannot take up in detail many of the points brought out, but as to classification, I agree with the term "cordal cancer," also the subglottic type. I don't like the term "extracordal." It is simply another term for extrinsic, which means that it is out of the larynx and does not definitely locate the growth. The term that I like to use, which was passed on to me by my father, is "epilaryngeal," which means that it is upon the larynx and its location is definitely mentioned — the epiglottis, aryepiglottic fold, arytenoid, and pyriform sinus. Knowing where the sites are makes a difference in the prognosis of these so-called external cordal or extrinsic or epilaryngeal types.

Then you also have a mixed type, and you do not know when you see a patient with a large mass in the larynx whether it is primarily intrinsic and is becoming extrinsic or laryngeal or vice versa.

The authors cite 58 cases in which the sites are not located and are what they term general. I think it would be well if they were broken down into the various sites.

In a paper presented before the Triological Society in 1951, I gave the sites of 344 cases operated on and their prognoses. The best prognoses were noted in the following order: vocal cords, epiglottis, epiglottis and tongue, arytenoids, ventricle, subglottic, pyriform sinus and aryepiglottic fold.

I agree with the authors that it would be an excellent idea for a committee to present a definite classification of nomenclature for all concerned. It would also help the radiologists to have the sites of cancer properly classified.

In a series of 525 cases, I found that approximately 8 per cent had leukoplakia which became malignant later, and 2 per cent were papillomas which became malignant later in life.

With regard to the relation of appearance and the actual extent of the carcinoma, it is generally known that only about one third of the growth is visible by direct or indirect examination of the larynx. It has been my experience that when in doubt as to when to do a laryngofissure or laryngectomy, I have always performed a laryngectomy, and after inspecting the larynx following removal, I have found that the lesion had extended either into the pre-epiglottic space, the subglottic space, or had invaded the opposite side by way of the anterior commissure.

Regarding the dearth of symptoms in the epilaryngeal or extracordal lesions, the spread of these tumors to regional lymph nodes occurs very

early in their growth, and extensive metastasis may be present before the primary tumor is manifest by symptoms, although there is one symptom that is fairly early and constant but, unfortunately, is overlooked both by the patient and the physician; that is, an abnormal sensation persistently felt in the throat. This can be brought out by a thorough and detailed history of the patient in the vast majority of cases. Also a lump in the side of the neck may be the first symptom that will take the patient to a doctor for consultation.

I agree with all that was said about the diagnostic aids. X-rays, planigrams, direct and indirect examination should all be employed before biopsy is taken, and the biopsy should be taken by the surgeon who is to operate.

Whatever may be our hopes about the discovery and treatment of malignant disease, of methods more specific and less harsh, surgical operations must be admitted to be the most useful and trustworthy means we now possess.

The surgeon who assumes the responsibility of obtaining a specimen for biopsy from a laryngeal tumor should be prepared and competent to treat the lesion. If a specimen of tissue is removed by someone else before the surgeon sees the patient, it is often difficult for the surgeon to determine the extent of the lesion. Once the diagnosis is made, it is necessary to decide, without prejudice, which treatment is best for the patient—surgery or irradiation. If irradiation is the choice, then the radiologist must be given full charge and left to adopt the technic best suited to the particular case. If surgery is chosen, then the surgeon's experience in this type of surgery must be taken into account. He must decide which is the best procedure to follow, taking into full consideration the age of the patient, the extent and situation of the growth.

The fact is that a case not suitable for operation is in all probability not a suitable one for irradiation other than as a palliative measure.

Here the experience of the surgeon in operating on cancer of the larynx and pharynx is of the utmost value, for it is from this experience that he, over a long period of time, has evaluated his failures and triumphs, and so has formulated in his mind the best surgical procedure adapted for each individual case.

In my series there were 150, or approximately 25 per cent, who used tobacco to excess. However, we have not found any increase in cancer of the larynx in females due to smoking, and they are doing a fair share of smoking today.

Hoarseness was noted in 75 per cent, yet we must not wait for hoarseness because carcinoma often is present without hoarseness. In a true subglottic cancer there will not be any hoarseness but there will be dyspnea. One of my patients, just returned from his honeymoon, complained of dyspnea. A large subglottic growth was found by the indirect method. Oftentimes patients present themselves for examination, realizing that something is wrong. They are reluctant to consult a doctor, mostly from fear of being told they have cancer; yet they themselves believe that they have. In my series nine months was the average time lost from the time the first symptom was noticed by the patient until a definite diagnosis was made.

ALDEN H. MILLER, M.D., Los Angeles, Calif.: Based on the observation of 116 patients in the past five years, Dr. Friedberg and Dr. Wallner have given us their conclusions regarding many of the pertinent problems in the diagnosis and treatment of carcinoma of the larynx. It is interesting and informative for all of us to compare our own experiences with theirs and to consider our own conclusions in the light of their deductions. For the most part my experiences with laryngeal carcinoma in private practice and on the laryngeal service and the Tumor Board of the Los Angeles County Hospital have been, with slight exceptions, the same as those of the authors and my concepts of etiology and choice of treatment are much the same as theirs. Unless considered too far distant by them, as an addition to their classification, based on the anatomic site of the lesion, I would suggest "postcricoid carcinoma," as we have seen more patients with the cancer limited to the postcricoid area and party wall than patients in whom the lesion was purely subglottic. In fact, we have not as yet seen a patient with carcinoma limited just to the subglottic area with no involvement of the vocal cords. This may be because we were not fortunate enough to see these patients at an early date before extension upwards had occurred. I like the authors' use and distinction of the terms "cordal" and "endolaryngeal."

A terrifically high incidence of smoking has been noted by our Tumor Board members not only in those patients with cordal cancer, especially those also demonstrating leukoplakia, but also in patients with malignancy of the lips, anterior tongue, floor of mouth, oral mucosa and the tonsils. However, no statistical comparison has been made with the incidence of smoking in the total population in the cancer age, although it has been reported as being about 75 per cent.

On our laryngeal service it has been noted that about half of our patients with cordal carcinoma have exhibited leukoplakia either clinically or by histopathologic examination. While all of these patients have been smokers, a few, especially women, have not smoked excessively. It has been of interest to me that in addition there

have been some patients who had completely stopped smoking on the first appearance of their hoarseness, some eight or nine months prior to their positive biopsy, and yet the leukoplakia had persisted until the time of their biopsy.

Lately, my greatest interest has been the increasing frequency of the finding of carcinoma in situ in the removal of what appeared to be simple leukoplakia, with no evidence of tumor of one or both vocal cords. I should like to ask the authors whether any such patients were encountered in their series and what was their treatment of these patients.

I, too, have observed that the greatest mistakes in clinically evaluating the extension of a malignancy have occurred in those patients with subglottic extension. I, therefore, also believe that the laryngofissure operation should rarely be considered in those patients in whom there seems to be subglottic extension.

Follow-up studies in our tumor registry have not revealed any unusual number of recurrences (less than 4 per cent) in cervical chains of glands following our laryngectomies. In fact, more of our recurrences have been about the tracheal stoma and on down into the mediastinum than in the cervical glands. Therefore, we still do very few so-called prophylactic neck dissections at the time of laryngectomy in those patients not giving clinical evidence of adenopathy.

Both our laryngeal service and x-ray department agree with the authors that surgery is the best treatment for subglottic carcinoma. However, we have had one patient with subglottic carcinoma in whom an attempt elsewhere at laryngofissure had been abandoned, following which the patient absolutely refused a laryngectomy and was therefore subsequently given x-ray treatment by the Coutard fractional method. He has been well seven years.

As the result of a continuing and surprisingly large number of dramatic cures, the first choice of the Tumor Board of the Los Angeles County Hospital for the treatment of extrinsic carcinoma of the larynx is x-ray irradiation. In our experience it is the extrinsic carcinomas which have produced the greatest amount of cervical gland metastasis.

DR. FRIEDBERG: Dr. Orton has objected to the use of the word "extracordal" and I must add that we are not wholly satisfied with the designation. There is, however, considerable variation in the nomenclature applied to laryngeal carcinoma, and we feel that the terms "cordal" and "extracordal" offer a sharp distinction between two forms which differ most with respect to treatment and prognosis. As we mentioned, it would be most gratifying if some international standardization could be brought forth in the field of laryngeal cancer.

We noted Dr. Orton's series of cases in which he subdivided extrinsic carcinoma into epiglottic, arytenoid, aryepiglottic fold and pyriform sinus lesions. We have followed a similar plan but did not include the figures in this report because of the unavailability of any long term results at this time.

Dr. Orton stated that only one third of the growth is ordinarily considered the visible portion of a neoplasm. For this reason we agree completely that laryngectomy is preferable in any borderline situation where the choice of surgery lies between laryngofissure and total extirpation.

The statistics quoted by Dr. Orton regarding the nine month interval between the appearance of symptoms and establishment of a definite diagnosis are roughly comparable to our observations.

In response to Dr. Miller, I should state that we have been accustomed to consider postcricoid tumors in the hypopharyngeal category.

As for carcinoma in situ, intraepithelial carcinoma, or preinvasive carcinoma — however one wishes to designate it — certainly more attention is being directed towards this entity in the current literature. In the first patient whose photographs were shown, we removed the entire vocal cord because of recurrent leukoplakia and found on serial sections that the pathologic basis for the recurrence was carcinoma in situ. There was no evidence of subepithelial invasion but there were definite signs of malignancy confined to the epithelial layer. We feel that one is justified in treating the lesion as carcinoma if there is any question of malignancy.

Figures on the efficacy of prophylactic neck dissections in laryngeal carcinoma are not available in sufficient numbers at this time. Dr. Ogura deserves credit for reviving interest in this concept, which seems to have been proposed originally by Sylvestre-Begnis. Dr. Clerf has long advocated neck dissection where any suspicion of metastatic nodes existed and has recently evidenced preference for an elective block dissection with laryngectomy in extensive lesions. At the present time the procedure of combined laryngectomy and neck dissection represents an effort to decrease the number of failures as a result of late cervical metastases, where surgery has adequately removed the primary tumor. We do not know what the final answer will be.

It was a privilege to hear Dr. Orton's and Dr. Miller's comments and we thank them for their stimulating discussion.

THE PATHOLOGY OF THE PHARYNGEAL RECESS, EUSTACHIAN TUBES, TONSILS AND ADENOIDS

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INTRODUCTION

IN THE uppermost corner of the nasopharynx, we find a deep pocket called the pharyngeal recess (recessus pharyngeus) or fossa of Rosenmüller (fig. 1). This pocket is more than a fossa; it is a hidden trap, first, because of its great depth, second, because of the mass of adenoid tissue that it contains, third, because malignancies arising in this location metastasize quickly, fourth, because cryptic infection is more common here than is generally supposed, and fifth, because a good many colds and attacks of nasopharyngitis begin here, and not a few patients point to this spot.

ADENOID TISSUE

The term "adenoid" comes from the word used by Hippocrates to mean "a gland" and the suffix meaning "like." It dates back to the time when anatomists, working with bare hands and not a little intuition, used such colorful terms as lymph glands and follicular glands in speaking of the lymphatic system.

The following quotation from Kolliker's manual of *Human Microscopical Anatomy*[19] is remarkably accurate, notwithstanding the fact that it was published in 1854—one hundred years ago:

> The pharynx contains two sorts of glands: (1) racemose mucous glands and (2) follicular (lymphoid) glands. The former have distinct apertures and abound more particularly in the upper portions of the pharynx, where they form a perfectly continuous layer upon the posterior wall, in the neighborhood of the pharyngeal opening of the eustachian tubes. Follicular glands, simple as well as compound, analogous to the tonsils, are met with in the vault of the pharynx. Here a glandular (adenoid) mass stretching from one eustachian opening to the other, and from one to four lines (2 to 8 mm.) thick, may constantly be met with; it is, upon the whole, smaller, but otherwise its structure resembles, in all essential respects, that of the tonsils. Besides this mass, whose largest sacculations (lobules) are situated in the middle of the roof of the pharynx, and in the recesses behind the eustachian apertures, and which, in aged persons, frequently present enlarged cavities, filled with puriform masses, there occur around the apertures of the tubes, and upon them, towards the choanae, on the posterior surface of the velum palati, and on the lateral walls of the pharynx, as far as the level of the epiglottis, more or less numerous, smaller and larger (lymphoid) follicles.

In the year 1868, Wilhelm Meyer called attention to the clinical importance of "adenoid vegetations," and thereafter the plural "adenoids" acquired a new meaning, namely, hyperplasia of nasopharyngeal lymphoid tissue. In this sense, the term "lateral adenoids" can be used when the pathology extends into the pharyngeal recesses, and the term "tubal adenoids" when the *lips* of the auditory tubes are involved (fig. 2).

LYMPHATIC NODULES

Lymphoid tissue stems from mesenchymal tissue, and in the beginning we see a scattering of lymphocytes called "diffuse lymphoid tissue." In time, the

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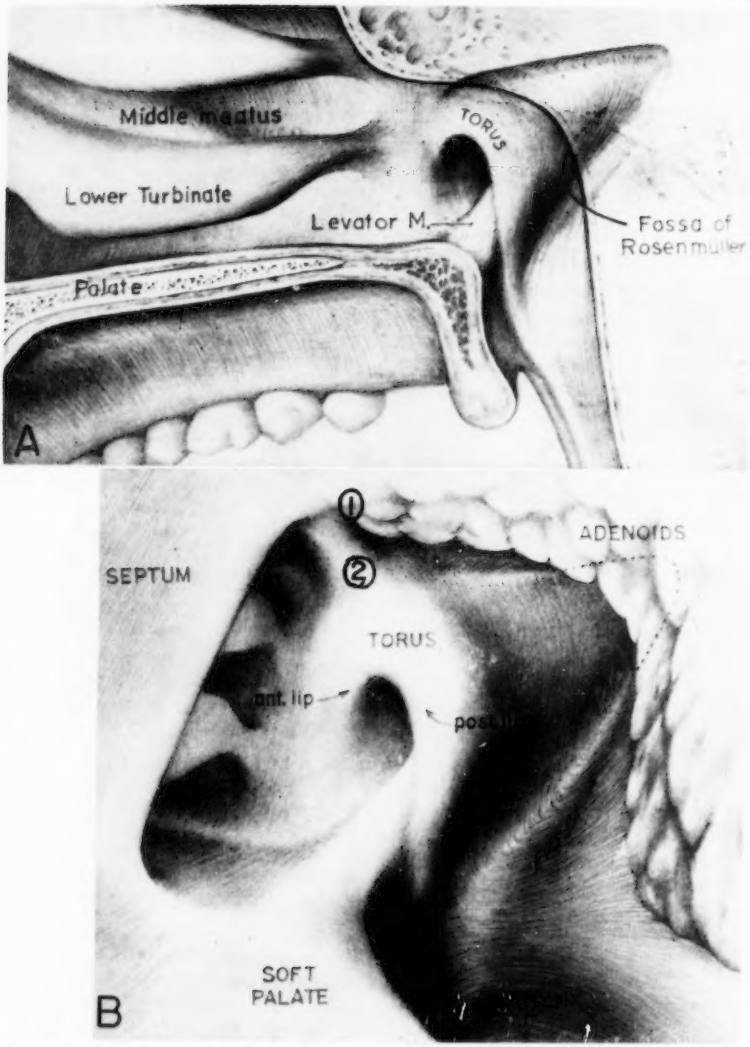

FIG. 1—The nasopharynx. (A) Conventional view of the ostium of the auditory tube, prominence of the torus tubarius, and, behind this landmark, the pharyngeal recess (fossa of Rosenmüller). Note the location of the levator veli palantini muscle in the tubal orifice. (B) The pharyngeal recess and location of (1) the nasopharyngeal pituitary gland and (2) the salpingonasal fold.

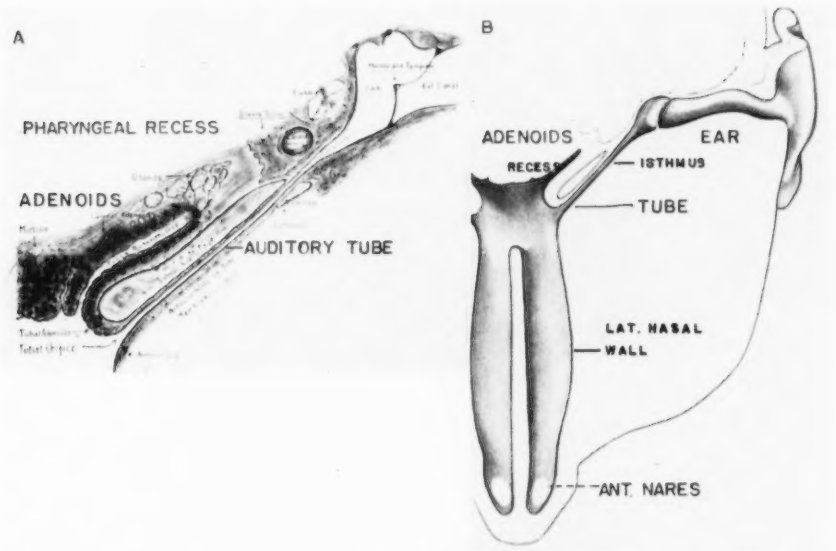

FIG. 2—A composite diagram showing the relationship of adenoids to the pharyngeal recess and auditory tube, drawn to scale from histologic sections. The circle is one centimeter in diameter. Note the depth of the fossa and oblique position of the tube. In discussing the eustachian tube most textbooks follow the terminology of Jacob Henle, an outstanding anatomist and histologist of a century ago. Owing to the oblique position of the tube he used the terms lateral and medial, where clinicians would find it more practical to use the terms anterior and posterior. His division of the tube into two parts (1) the osseous (tympanic) portion, and (2) the cartilaginous (pharyngeal) portion is well known. The isthmus acts as a dividing line between these two portions, and at this coupling the tube is always narrow and sometimes slightly bent or kinked, creating a natural obstruction which frequently is mistaken for a stricture.

cells increase in number and eventually form primary lymphatic nodules (congregations of small lymphocytes) in the bed of diffuse lymphoid tissue. In tonsillar tissue, at the age of 9, 10, or 11 months, secondary lymphatic nodules, or follicles, begin to appear.[1] They contain in their center, which is rather pale and soft, (1) many medium-sized lymphocytes, (2) a few large lymphocytes, (3) large reticular cells with indistinct cytoplasmic outlines, (4) histiocytes, and (5) macrophages (fig. 3).

The follicles are about half a millimeter in diameter and when hyperplastic, they become large and rupture easily, which prompted Kolliker to remark, in speaking of tonsils, ". . . in the course of inflammation—the contents of the follicles appear to alter, the follicles themselves becoming distended and finally bursting," an observation that anyone who performs a tonsillectomy can easily confirm.

Ehrlich's blood stains became available toward the end of the nineteenth century, and within a few years the origin and function of lymphoid tissue became more apparent. In 1885 histologists decided to abandon the term "follicular glands."

In 1884, Flemming called the secondary nodules "germinal centers" because he saw many mitotic figures in them. This narrow conception of their function is now out of date. In fact, modern pathologists have a broader conception of their function, and many call them "reaction centers."

Under perfectly normal conditions they are not present at the time of birth.[4] According to Ehrich,[5] they appear after an infection or a reac-

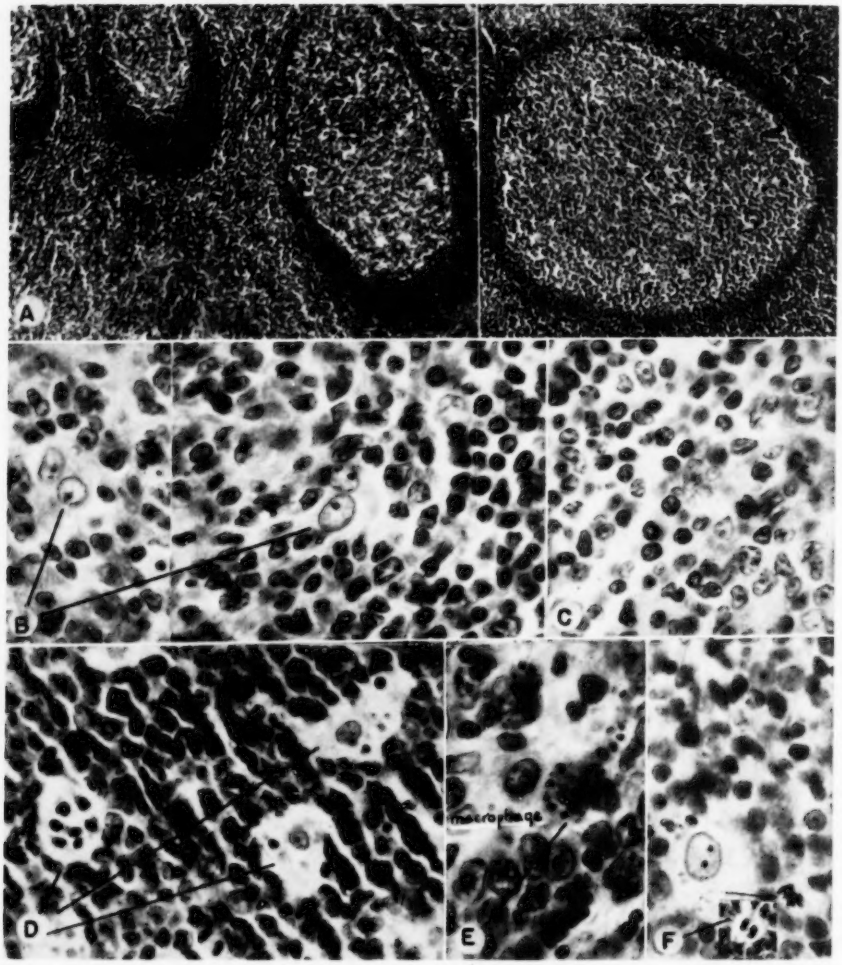

FIG. 3—Adenoid (tonsillar) tissue. (A) Lymphoid follicles showing germinal (reaction) centers surrounded by signet ring of lymphocytes (x70). (B) Cytology of reaction centers. Histiocytes have large nuclei and indistinct cytoplasmic outlines. (C) Medium-sized lymphocytes. (D) Histiocytes containing cell fragments. (These fixed macrophages eat bacteria and also lymphocytes.) (E) Macrophage containing many cell fragments after radium irradiation. (The surrounding cells are hydropic.) (F) Mitotic figure in a medium-sized lymphocyte (x700).

tion of some sort has taken place. Similar conclusions were reached by Glimstedt (Drinker and Yoffey[7]), who succeeded in raising guinea pigs in an environment from which all bacteria were excluded; Wiseman,[42] who administered foreign proteins parenterally and produced an *allergic hyperplasia* of the follicles, and Miller,[23] who studied abdominal lymph nodes.

In newborn infants, normal tonsils and adenoids are made up of (1) primary lymphoid nodules and (2) diffuse lymphoid tissue. The secondary follicles (germinal, or reaction centers) that characterize tonsillar tissue and give it a distinctive name, apparently are due to inflammation and a number of other influences that come into play later in life. They are not as simple as they look, and by histological methods alone, we barely can make out what is going on, recognizing only a few alterations, for example: (1) enlarged and hyperplastic follicles, (2) follicles with many lymphoblastic cells, (3) follicles with many macrophages, e.g., following irradiation, (4) follicles with involutionary changes, and (5) necrotic follicles due to infection. The reaction centers can produce lymphocytes and they can destroy them, and when they outlive their usefulness, they fade away as mysteriously as they come.

New areas of lymphocytopoiesis and/or follicles can appear almost anywhere that we find primitive reticular (mesenchymal) tissue. The primitive reticular cells generate medium-sized lymphocytes (lymphoblasts), macrophages, and other cells, as well as the reticulum, or stroma, that supports them.

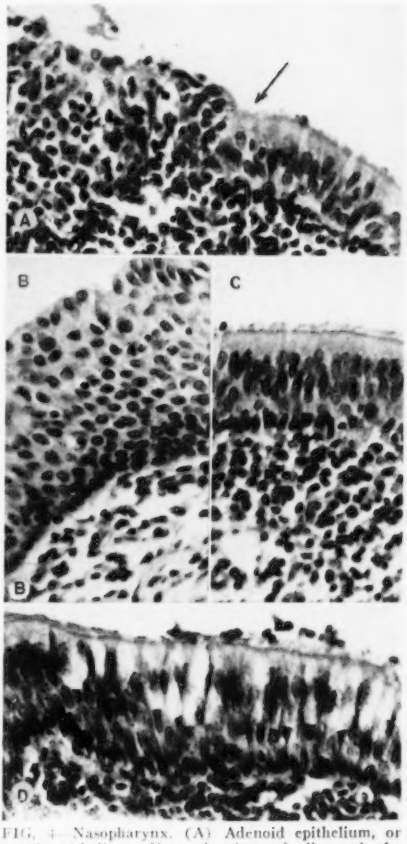

FIG. 4.—Nasopharynx. (A) Adenoid epithelium, or lymphoepithelium. Note the intermingling of the lymphocytes and ciliated columnar cells. (B) Transitional epithelium, or hyperplastic epithelium. (C) Normal pseudostratified ciliated columnar epithelium (×600). (D) Goblet cells.

HISTOLOGY OF THE NASOPHARYNX

The mucosa of the nasopharynx is a direct continuation of the nasal mucous membrane. Beneath the tunica propria and sometimes not clearly separable from it, we find the submucosa. The lymphoid layer always is superficial and confined to the tunica propria. (In dissection of the faucial tonsils, for example, the tonsil and its capsule [L., *little box*], can be stripped away en masse and separated from the submucosa.)

The nasopharynx (epipharynx), as Kolliker stated, contains many mucous and serous glands. The seromucous acini lie below the lymphoid tissue layer, in a strong supporting stroma. But the excretory ducts, which sometimes are wide enough to be called crypts, pass through the soft substance of the lym-

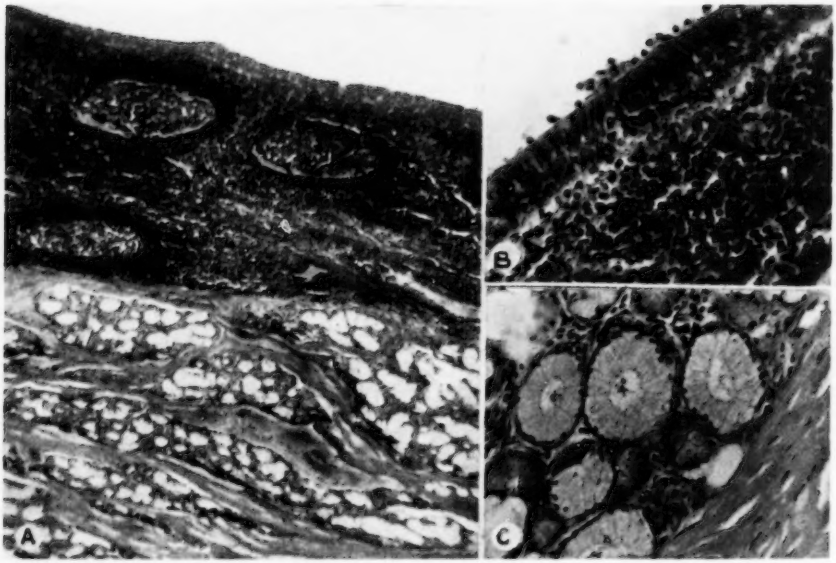

FIG. 5—Mucous membrane lining the pharyngeal recess. (A) Note the layers of the tunica propria, (1) the lymphoid tissue layer (the superficial [reticular] layer) and (2) the glandular layer (the deeper collagenous connective tissue stroma) (x70). The line of demarcation between these layers is a sharp one until we attempt to separate them surgically. Conserve the glands, they lubricate the nasopharynx. (B) Note the lack of a true basement membrane. The epithelium sits directly on reticular (lymphoid) tissue (x300). (C) Mucous and serous glands (x300).

phoid layer and, in this vulnerable location, have a tendency to become dilated or cystic.

THE EPITHELIUM

The epithelium of the nasopharynx normally is of the pseudostratified *ciliated columnar type* and spreads over the adenoids and into their crypts. Like the epithelium of the nasal passages, it contains mucous (goblet) cells and may undergo a physiologic metaplasia, i.e., adapt itself to air currents[28] and other environmental changes by becoming thicker or thinner. Needless to add, we find pathologic metaplasia with inflammation and injury.

The transition to stratified squamous epithelium, as might be expected, takes place at the junction of the epipharynx and the mesopharynx. The *transitional epithelium* that appears in this more or less variable zone is unciliated and stratified, i.e., composed mainly of columnar cells.

CONNECTIVE TISSUE

In dealing with connective tissue we must reckon with (1) its intercellular substances, (2) its fibers, and (3) its cells. The reticulum fibrils that support the lymphocytes can be demonstrated with special stains. The collagen fibers, being of heavier construction and more cohesive, can be seen in ordinary section (fig. 5). Hyaluronic acid, a mucopolysaccharide (intercellular substance) can be demonstrated with special stains. Hyaluronidase, the enzyme or spreading factor that lowers its viscosity, is a new agent that we appreciate because it enhances the infiltration of procaine. I find it useful in removing adenoids of adults under local anesthesia.

FIG. 6—The lobules, clefts, stroma, trabeculae and follicles of a typical pharyngeal tonsil (median adenoid mass). Compare the superficial layer of follicles with those shown in the next picture (fig. 7). (x8)

Reticulum fibrils are anatomically continuous with collagen fibers and sometimes are designated as pre-collagenic. When they mature, they become collagen fibers, e.g., after involution of the lymphoid tissue.

The role of intercellular substances in collagen diseases is just beginning to unfold, even though we have known for a long time that collagen gets its name from the fact that it is the source of glue (Gr., *colla*). In chronic tonsillitis, nature glues the broken parts with collagen, creating the scar tissue of fibrotic tonsillitis.

THE PHARYNGEAL TONSIL

The pharyngeal tonsil, or median mass of adenoids, sometimes reaches a thickness of 1 cm. and a width of 2.5 cm., the distance between the two eustachian tubes. The size, of course, varies with the age of the individual and amount of hyperplasia, for which reasons we have three different sizes of adenotomes. The lobules hang from the vault of the nasopharynx, separated by clefts that are fairly deep—so deep in fact that the lobules often separate like the leaves of a book and fall out of our adenotomes. They are supported by fibrous septa, offshoots of the underlying fibrous stroma, and do not have a true capsule around them.

The glands lie in the depths of the stroma, and wherever we have crypts and ducts, we occasionally have retention cysts, the cystic material being epithelial (goblet cell) and glandular in origin. When the cysts become infected, they can be mistaken for Tornwaldt's disease, or pharyngeal bursitis, a condition that for all practical purposes amounts to the same thing. This type of pathology can occur at any age, is more common in young adults, and sometimes appears in the pharyngeal recess. Histologically, the cysts are lined with respiratory epithelium, which may (a) undergo metaplasia or (b) be lost entirely. The jellylike contents may be burdened with cellular debris and cholesterin crystals, or, when an abscess occurs, purulent material. Kolliker[19] mentioned these "puriform masses" long before Tornwaldt, Waldeyer, Luschka or Meyer came upon the scene.

Tonsils and Adenoids During Infancy

At the time of birth, our tonsils and adenoids are as near normal as they ever

will be; they are small, the crypts are comparatively clean, and the epithelium is intact.

In six months or so, they increase in size and show signs of follicular hyperplasia, the degree being determined by a number of factors — infection, allergy, immunity, a host of defense mechanisms, homeostasis, etc.

INVOLUTION OF LYMPHOID TISSUE

In children the lymphatic tissue of the entire body is more luxuriant than it is in adults, owing presumably to hormonal influences. Between the ages of 6 and 16 years, or onset of puberty, a new set of hormones takes over, and the adenoids are among the first to show a tendency to involution and atrophy. By the time we reach forty, the follicles have faded from view, leaving some diffuse lymphoid tissue behind, along with whatever pathology may have developed during the years in the epithelium, parenchyma, stroma, and crypts. We have known for a long time that an adrenal insufficiency will cause delayed involution of lymphoid tissue, which should serve to remind us that the size of tonsils and adenoids is not entirely a matter of infection.

FUNCTION

On one hand, tonsils and adenoids produce lymphocytes, and on the other, they destroy them. Even in health, their crypts harbor a subclinical state of infection. Many viruses and bacteria presumably reach the reaction centers, where they are put through the mill of macrophages for purposes of antibody formation.

Antibody formation, it is said, begins with macrophages and is completed by lymphocytes. Lymphocytes have a short life cycle, i.e., they remain in the blood about 12 hours.[7] When they undergo dissolution their cytoplasm yields gamma globulin. Cortisone hastens their disappearance from the blood and brings about their dissolution, much in the same manner that radiation does. In short, the pituitary-adrenal apparatus influences the size and development of lymphoid tissue.[41]

Nasopharyngeal Pituitary Gland

A small accessory pituitary gland can be found in the region of the vomerosphenoidal articulation, i.e., foremost part of the nasopharyngeal tonsil (mucosa of the nasopharyngeal vault). It is composed of cells exactly like those of the anterior lobe of the pituitary gland and marks the anlage (Rathke's pouch).

Melchionna and Moore[22] found the pharyngeal pituitary gland in fifty-one of the fifty-four cadavers that they examined (95 per cent) and, in a few cases, observed definite vacuolization of the cells, which is a sign of function and secretory activity.

Somatotropic hormones (STH) and adrenocorticotropic hormone (ACTH) are so potent that a few molecules in the right place can produce some profound effects.

What connection, if any, is there between the growth and involution of adenoids and the pharyngeal pituitary gland? Until we have an answer, one way or the other, I believe it is wise, in performing adenoidectomies, to proceed with caution and permit a strip of tissue at least 6 mm. in width and about 10 mm. in length to remain intact in the area occupied by this gland. Fortunately, the gland lies just behind the nasal septum in an area a little beyond the reach of the adenotome (fig. 1).

STATUS THYMICOLYMPHATICUS

About twenty-five years ago, I performed an autopsy on a cherubic boy who went to school one day and expired there for no apparent reason. Because the symptoms resembled laryngeal diphtheria, the officials quarantined the classroom and everyone in it. They ordered an immediate autopsy and this is what impressed me: (1) hyperplasia of all lymphoid tissue organs, (2) a very small aorta, and (3) the very small size of the adrenal glands.

Status lymphaticus is a constitutional defect in which an inadequacy of the suprarenal glands and the autonomic nervous system probably explain the notorious tendency to sudden death. It is characterized by delayed involution, or hyperplasia, of the thymus, tonsils, adenoids and lymph glands; underdevelopment of the gonadal and cardiovascular systems; certain peculiarities of external configuration (a pale skin and excessive de-

velopment of subcutaneous fat pads); and diminished resistance to physical, bacterial, allergic, toxic and psychic stress.

Marine[20] expressed the opinion that the lymphatic hyperplasias of childhood are manifestations of a functional underdevelopment of the adrenal cortex.

What are the practical implications? (1) Extremely large tonsils in an angelic looking child invite disaster and despair. (2) Will Cortisone or some related hormones prevent death in such a crisis?

HYPERPLASIA

Hyperplasia of lymphoid tissue depends upon a great many factors, and, in general, may be divided into three broad categories:

1. Endocrine, e.g., status lymphaticus
2. Inflammatory hyperplasia, e.g., infection or allergy
3. Neoplastic hyperplasia, e.g., the lymphomas

In hyperplastic tissues we find an increase in the number of cellular elements and, as a rule, an associated abnormality of function e.g., hyperplastic rhinitis, hyperplastic (toxic) thyroid. Hyperplastic tonsils and adenoids sometimes become so large as to interfere with respiration and deglutition, which certainly is an abnormal function. Moreover, a slight infection provokes an inflammatory reaction that often is out of all proportion to the cause. The swelling and inflammatory reaction may be reduced with antibiotics and Cortisone, but so far the reports dealing with the use of ACTH and Cortisone in nasal polyps and nasopharyngeal lymphoid tissue do not pretend that the cellular hyperplasia is reduced to any great extent.[2,4,35]

APPLIED PATHOLOGY

Surgery puts a patient in bed and his disease in a bottle. A pathologist cannot look into a bottle and state definitely whether or not the adenoids are pathologic. With a microscope he may observe the follicular hyperplasia, inflammatory exudate, and at times cysts, but he cannot see the clinical picture—the blocked postnasal passages, disease of the auditory tube and middle ear, or deafness.

With every acute sore throat, whether due to streptococci or a virus, scarlet fever or infectious mononucleosis, the nasopharynx and adenoids become infected. In *acute inflammation*, the adenoids become red and angry, the crypts fill with purulent material, epithelial debris, lymphocytes, bacteria, and, in the more severe cases, fibrin.

The epithelium suffers injury; polymorphonuclear leukocytes and plasma cells appear in the stroma and subepithelial region; the blood vessels show active hyperemia, and their endothelial cells become swollen. The reaction centers join in the battle; their macrophages engulf the bacteria and sometimes break down, which, in severe infections, causes abscesses in the follicles.

In *chronic hyperplastic adenoids* we find a proliferation of lymphoblasts and reticulum cells, and enlargement of the reaction centers. This is the salient feature of the pathology. Needless to add, various degrees of infection, either acute or chronic, may complicate the picture.

THE ADENOID PROBLEM

Inasmuch as there is some difference of opinion regarding the "regrowth of adenoids," and not a little speculation concerning their proper management, it may be well to consider the effects of irradiation of adenoid tissues at this time.

The following cases, chosen from my practice, illustrate the adenoid problem and, at the same time, demonstrate the clinical and histologic effects of irradiation.

Case 1. M. D., 7 years of age, complained of chronic nasal obstruction and gave a history of an alleged removal of adenoids at the age of 6 years by a general practitioner. When this failed to help him, he received 30 weeks of aureomycin therapy and, finally, a diagnosis of nasal allergy from his pediatrician.

Examination. After shrinking the nasal mucous membranes and removing a puddle of thick mucus, one could plainly see, by anterior rhinoscopy, adenoids large enough to interfere with elevation of the soft palate. The nasal mucosa had the pale appearance of allergy, but the nasal smears were devoid of eosinophils. Roentgeno-

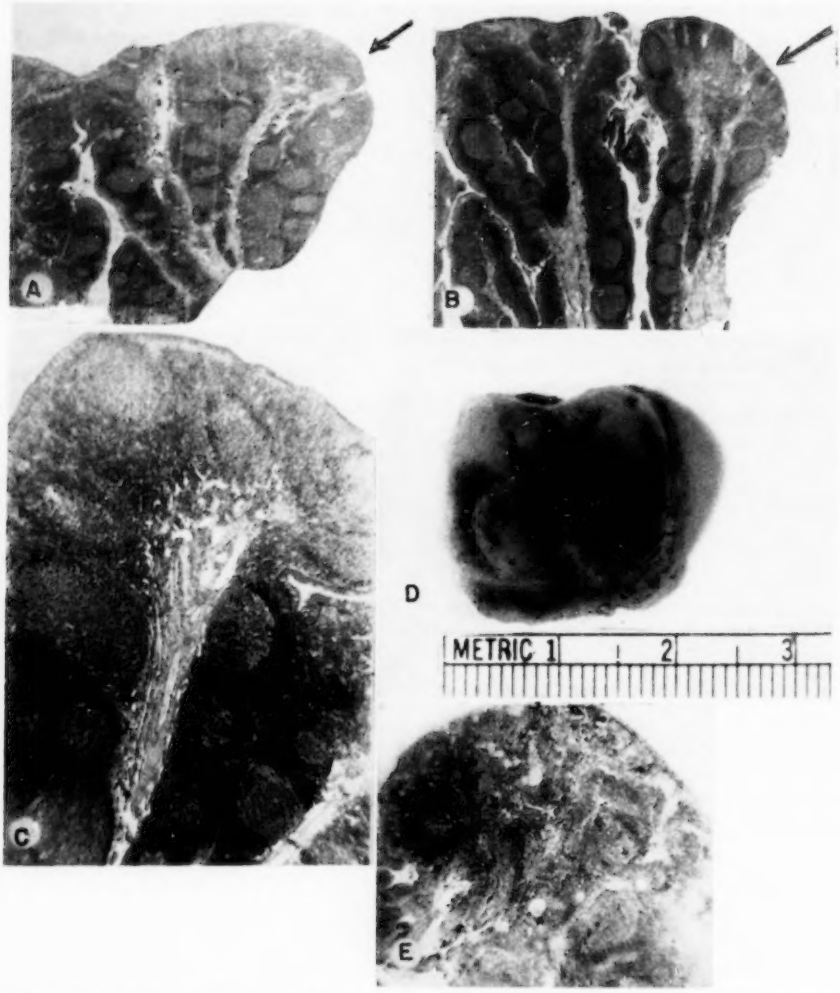

FIG. 7—(A) Radium irradiation has "burned out" the signet rings of the follicles. The effect is more noticeable in the first two centimeters of tissue (upper right hand corner). (B) Effect is less in this lobule (x4). (C) The more superficial follicles are poorly outlined (x10). (A, B, and C are from case 1.) (D) Gross specimen of "regrowth of adenoids" from another case. Note the markings and crypts of an adenoid tag and not a regrowth. The adenoidectomy should have been done properly the first time. (E) Roentgen therapy. (Case 2) The effects are widespread and deep. All of the follicles are poorly outlined and identified with difficulty. Much scar tissue is present (x5).

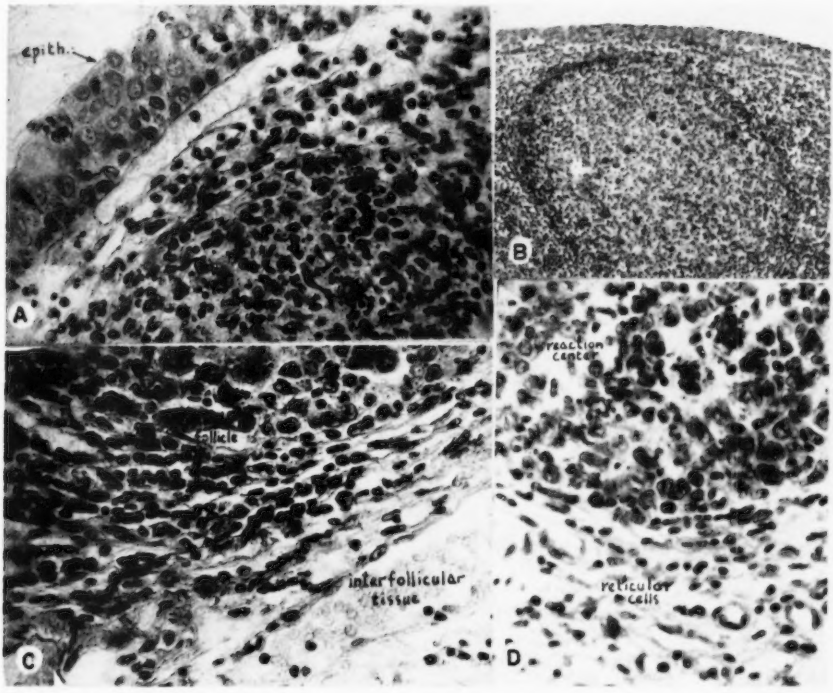

FIG. 8—(A) The lymphocytes have almost disappeared from the subepithelial region, revealing the reticulum (x300). (B) Follicle showing loss of lymphocytic ring (x70). (C) Rim of reaction center showing reticulum (x300). (D) Macrophages in reaction center (above) and loss of lymphocytic ring (below). (x300) (Case 1)

grams revealed a catarrhal sinusitis, which is not unusual when a postnasal obstruction interferes with ventilation and drainage. He obviously needed a proper adenoidectomy.

Irradiation. The day before surgery, a 50 mg. monel radium applicator was applied to the adenoids, each side receiving ten minutes of irradiation. (The irradiation in this case was given for histologic purposes.)

Adenoidectomy. Eighteen hours later, I removed a large mass that had all the anatomical markings (crypts, etc.) of a pharyngeal tonsil rather than "regrowth" of lymphoid tissue.

The patient made a rapid and uneventful recovery and has no symptoms or signs of "nasal allergy," snoring, mouth breathing, nasal discharge, or sinusitis since the time of surgery.

Histology

The histologic effects of the irradiation may be summed up as follows:

1. The superficial layer of tissue, a zone about 2 mm. in thickness, takes a pale stain due to destruction of lymphocytes, particularly the small, dark-staining mature ones that appear in the interfollicular tissue and signet rings (fig. 7A).

2. The deeper follicles show less reaction, which is consistent with Braestrup's figures.[5]

3. The stroma stands out because it contains fewer lymphocytes (fig. 8).

4. The germinal centers can be recognized, but not as easily as before because the ring of lymphocytes that frames them is lost.

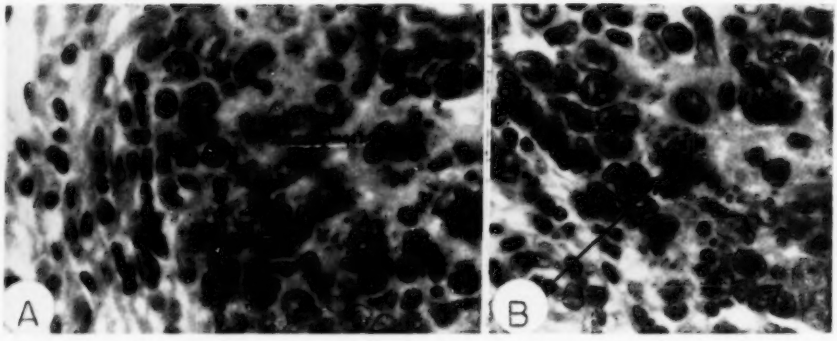

FIG. 9—(A) Margin of reaction center. (B) The center itself, showing swelling, fragmentation of cells, and macrophage filled with cell fragments (x700).

5. The germinal or reaction centers are a scene of great activity. The macrophages are filled with dark-staining particles, the fragments of broken up lymphocytes (fig. 9).
6. The chief cytologic effects are the pyknosis, karyorrhexis, injury and dissolution of the mature lymphocytes.

Case 2. A boy, 6½ years of age, with all of the symptoms and signs of chronic tubal obstruction, persistent deafness and otitis media, had an adenoidectomy at the age of 4 years. He continued to have ear trouble, and at 5½ years was given roentgen therapy on the advice of a pediatrician, receiving a total of 537 r to the level of the eustachian tubes. In spite of this broadside, from ear to ear, the deafness continued, and, ten months later, audiograms showed an average loss of 43 decibels.

Nasopharyngoscopic examination revealed a large mass of red adenoids blocking the tubes. A careful second adenoidectomy was performed and and a fibrotic mass of adenoids that weighed 1.3 gm. was removed. A postoperative audiogram ten days later showed a 10 decibel loss of hearing acuity. He has been asymptomatic and his hearing has been good for the past three years.

Histology

The histologic examination of adenoids may be summed up as follows:

1. A marked reduction in the number of lymphocytes throughout the entire thickness of the adenoid (fig. 7E).
2. Much scarlike connective tissue, i.e., keloids resulting from previous inflammation, surgery and irradiation.
3. The "reaction centers" contain many macrophages and phagocytosed debris.
4. The entire mass is uniformly "burned out," and the effects of x-ray irradiation are deep and widespread.

Comments Concerning Irradiation

The reaction to irradiation, it is said, begins within one hour and is intense at the end of eight hours, continuing on for five or six days, during which time mitosis is at a standstill. At a later time, mitosis begins again, but at a slower pace.

The connective tissue in *fibrotic adenoids* (keloids) remains standing, like the girders of a burned out building, and in some cases forms an obstruction that cannot be resolved with irradiation. For this reason, and others too numerous to mention, there is no substi-

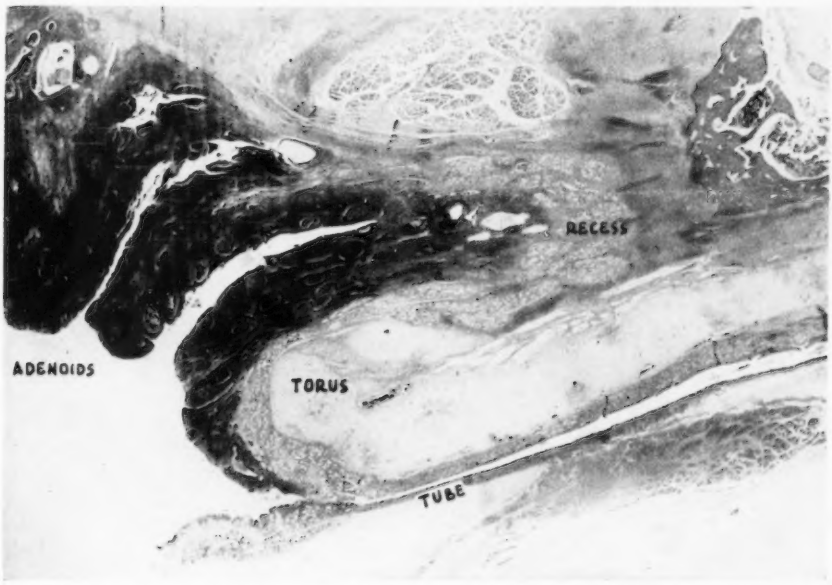

FIG. 10—Adult nasopharynx. Horizontal section showing the adenoids, the pharyngeal recess, and the eustachian tube. The adenoid tissue extends to the tubal orifice but ends at the entrance to the tube. There is no lymphoid tissue within the tube itself (x7). (AFIP no. 82362) (Courtesy E. P. Fowler, Jr.[11])

tute for a careful and complete surgical adenoidectomy.

When irradiation is applied to adrenalectomized animals, deficient in Cortisone and other adrenal secretions, larger doses of irradiation are needed to accomplish the same results, and the rate of regeneration of lymphocytes is more rapid. This may account for some of the unpredictable results seen clinically.

ADENOIDS IN ADULTS

Notwithstanding the fact that adenoids were first discovered in adults, they loom so large in children that very few people think of them as a disease of adults (fig. 10). For this reason, hidden or cryptic nasopharyngeal infection often is overlooked in adults. Buried adenoids in the pharyngeal recess are just as bad as buried faucial tonsils, the only difference being that one can be seen with a tongue blade and flashlight and is easier to get at than the other. The problem is an old one and many otolaryngologists have discussed it from time to time. Theobald[39] has mentioned the pathology, and, more recently, tabulated its clinical symptoms.

In 1908, Jervey[16] remarked that bands, webs and lymphoid tissue, sometimes quite filling the fossa, especially in adults, create an illusion of a very shallow or absent fossa, and that the fossa is never absent or very shallow in the adult. He also described the digital technic (finger surgery) for breaking up the adhesions, a procedure that I often use in my office practice.

In 1911, Holmes[14] taught us how to use the electric nasopharyngoscope. As a consequence of his rich experience with it, he remarked that much pathologic tissue often remains after an adenoidectomy, and that ". . . it is surprising to find how frequently small amounts of adenoid tissue in the lateral fossa and upon the cushion

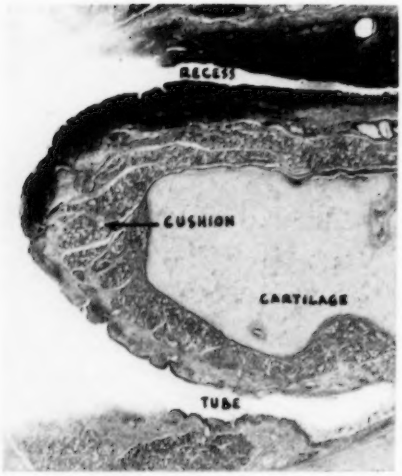

FIG. 11—The eustachian cushion (x7). The lymphoid layer is superficial and confined to the posterior aspect of the cushion, which is fairly normal even in adults. This specimen and the one in figure 10 are from aviators. (AFIP no. 83331)

of the tube cause chronic congestion of the whole tube."[15]

Yankauer[16] concluded that adenoids along the salpingonasal fold can cause nasal obstruction; he remarked that our adenotomes slip between the tubal eminences and remove the central mass but miss the lateral extensions. Reeves and Brill[20] recently expressed a similar opinion.

The Pharyngeal Recess

The full depth of the recess (fig. 1) is about an inch. Unfortunately few anatomy books offer a clear picture of it, which prompted Prentiss[27] to remark, ". . . the texts are curiously obscure." According to this author, the fossa is derived along with the eustachian tube, from the first visceral pouch. He described it as a contracted cone, and mentioned the sinus of Morgagni, a defect in the pharyngeal aponeurosis.

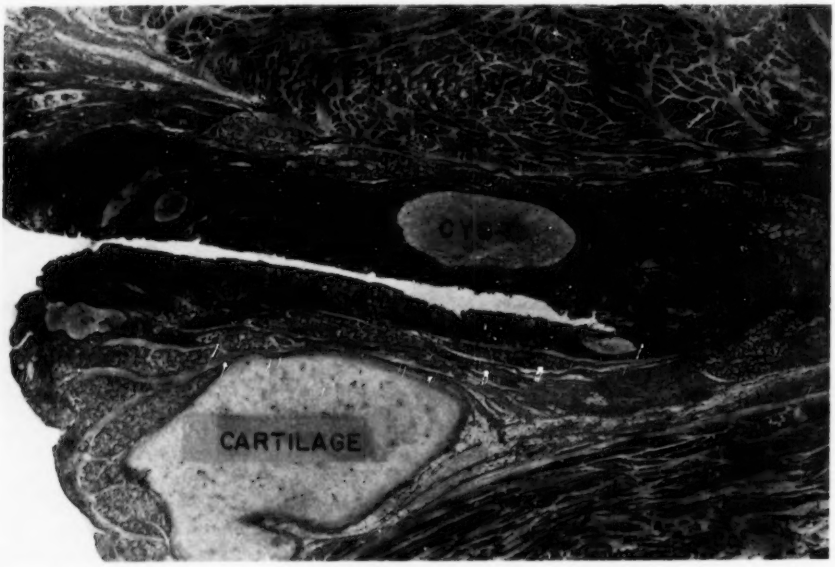

FIG. 12—Pharyngeal recess (x7). From above downward, pharyngeal wall, adenoid tissue in the recess, retention cysts, slit-like lumen of the recess, tubal wall. Note the great depth of the fossa and large amount of lymphoid tissue on the tubal cartilage. This is from an adult. (AFIP no. 83326)

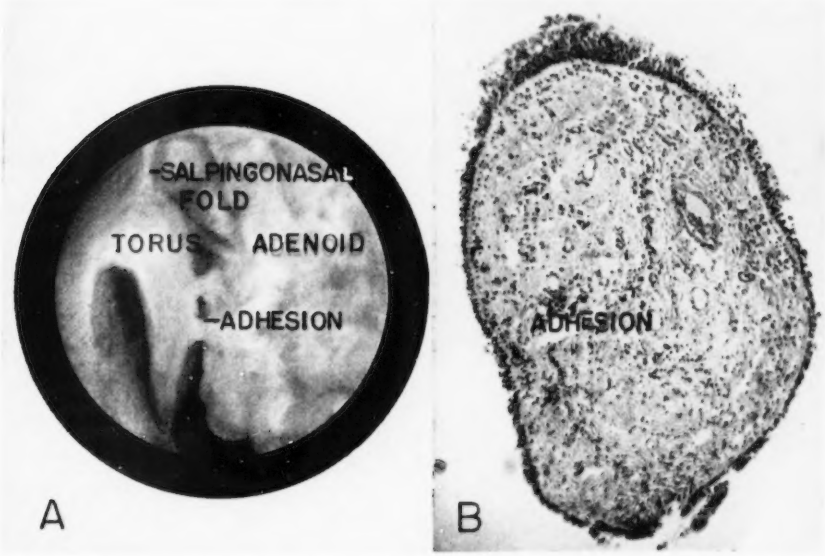

FIG. 13—Adhesion in the recess. (A) As reviewed through a nasopharyngoscope. (B) In histologic cross section showing hyperplastic epithelium and white fibrous scar tissue. As a rule the adhesions are formed during periods of acute inflammation in which the epithelial surfaces ulcerate and raw surfaces unite.

The walls are made up of the following histological layers: (1) ciliated columnar epithelium, (2) a layer of lymphoid tissue, (3) a glandular layer, and (4) submucosal connective tissue and muscular layers.

EXAMINATION

To examine the recess, one must use the electric nasopharyngoscope. This simple office procedure reveals details that no other device can equal. With a few minor variations, I follow the technic outlined by Holmes[4] years ago.

Procedure

1. Shrink the nose with a vasoconstrictor spray.

2. Swab the inferior meatus with a topical anesthetic.

3. Pass the nasopharyngoscope along the floor of the nose, lens upward to keep it clean.

4. Rotate the lens to locate the posterior border of the septum, then rotate the lens to examine the

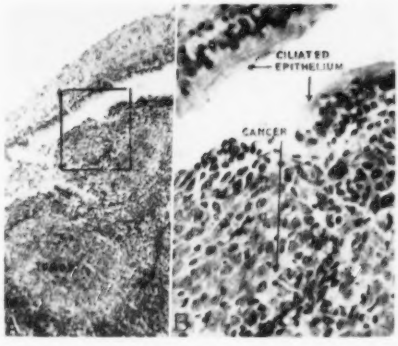

FIG. 14—Lymphoepithelioma. Ring punch biopsy through a Yankauer speculum. The primary lesion in the pharyngeal recess was no bigger than a pinhead. The first symptom, cervical adenopathy. Metastasis in this location is rapid because of the lymphatic trunks that pass through the recess into the neck. (A) At first glance the nest of tumor cells looks like a follicle (x70). (B) Higher magnification showing the undifferentiated nature of the tumor cells (x300). They have poor outlines. Note the loss of continuity in the overlying ciliated columnar epithelium.

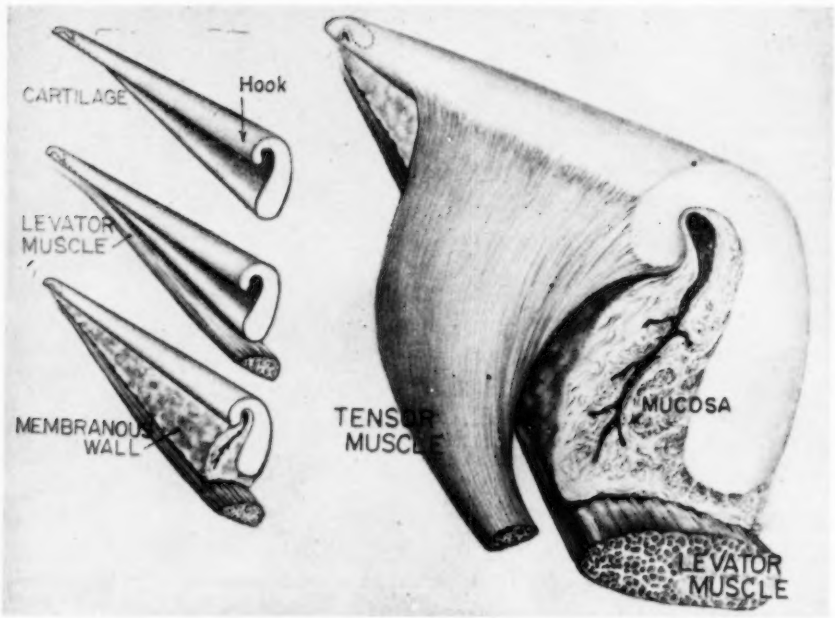

FIG. 15—Diagram of the cartilaginous portion of the auditory tube. The anterior wall is membranous. The posterior wall is the cartilaginous one. The mucous membrane is thick and contains longitudinal folds. (A) The folds help close the tube. (B) When the tube opens, the tensor muscle acts as an abductor, while the levator muscle elevates the cartilage and displaces it backward into the fossa. This movement is arrested by obstacles in the pharyngeal recess.

torus tubarius, the nasopharyngeal fold, mouth of the tube, the pharyngeal recess, and the vault of the nasopharynx.

5. Determine the motility of the posterior lip of the tube.

A careful routine will reveal the presence of pathology; a hasty one will miss it. As stated in a previous publication,[36] we must use every known device to examine the nasopharynx properly.

Interpretation of findings. Normally, the fossa is clean, the cushion of the tube is smooth, and the posterior lip moves freely. A fold of mucous membrane, called the salpingonasal fold or nasopharyngeal fold, at the apex of the torus tubarius (fig. 1) is a landmark worth noting. In epipharyngitis, the reddening begins at this fold and extends backward into the fossa.

The following changes are significant: (1) reddening of the mucosa, (2) redundant tissues, pale or red, (3) granular thickening of the mucous membrane, (4) stagnation of mucus, and (5) an insignificant "granulation" that bleeds when touched, which might be a lymphoepithelioma.

Function. The recess *allows room for expansion of the eustachian tube,* and it is a scene of great activity when the tube opens.

Owing to tissue turgor, it is not as spacious during life as it is in dried anatomical preparations, but even so, when it seems shallow or obliterated, one must

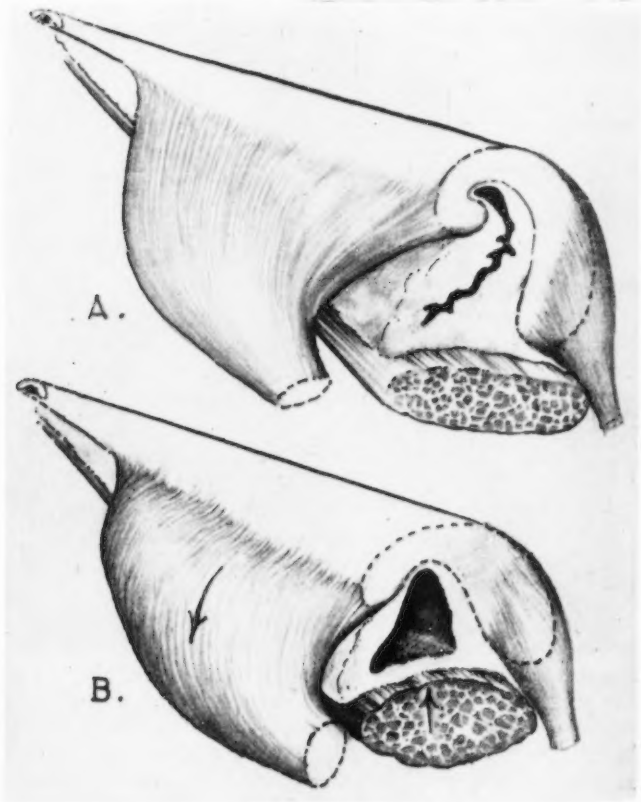

FIG. 15.—Second part.

determine the cause. Adenoids in the recess hamper the tube and rob it of its "elbow room." Adhesions sometimes do the same thing and often convert the recess into a quagmire of pseudocystic pockets. The secretions that accumulate in the recess often cause a chronic postnasal drip. In determining the cause of this complaint, I first rule out nasal allergy, then sinusitis, and always chronic epipharyngitis. As might be expected, these conditions often coexist, but any one alone may account for the chronic perplexing catarrhs that plague our patients.

The Pharyngeal Portion of the Auditory Tube

The pharyngeal (cartilaginous) portion of the eustachian tube is so named because it belongs to the pharynx.[32,44] About an inch in length, it lies in the pharyngeal recess, and a large part of the cartilaginous plate (posterior wall) can be palpated in this location.

PHYSIOLOGY

When the tube opens, the anterior lip becomes tense, the posterior lip swings upward, backward, and at the same time, toward the midline. Meanwhile the recess contracts to a mere slit. Because

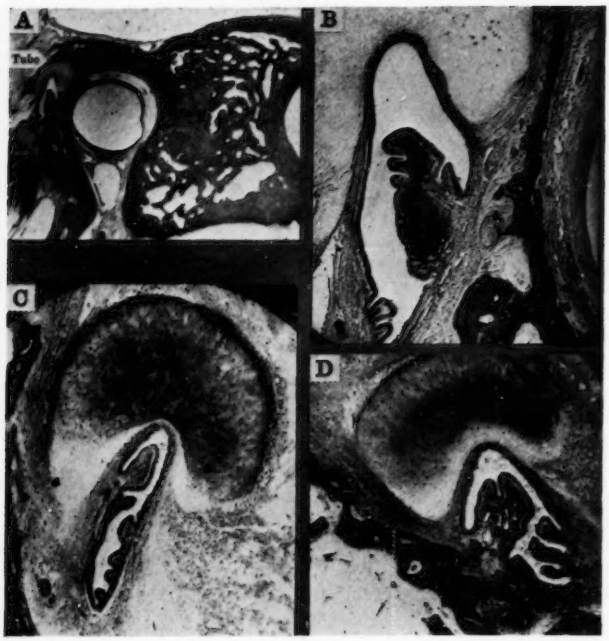

FIG. 16.—Auditory tube, cross section near the isthmus. (A) Petrous portion of the temporal bone (×2). The auditory tube is shown in its relation to the carotid artery at this point. (B) Higher magnification showing the hook of Henle above. The dark mass in the lumen of the tube is a dense round cell infiltration in the folds of the tubal mucosa. From a case of chronic suppurative otitis media. (C) Folds in the mucosa of an infant. (D) The lumen of the tube of another infant. In both instances round cell infiltrations appear in the subepithelial region. The folds in (D) are hyperplastic. Specimens from the Johns Hopkins Otological Research Laboratory. (Semenov[21])

of its springlike action, mucosal folds, fat pads, and slight spiral, the cartilage tends to close the tube.[22] The tubal muscles open the spring, in keeping with the hinge of the cartilage (fig. 15).

The *levator* veli palatini lies along the floor of the tube. Its cylindrical belly produces a bulge that rises into view as the tube opens (fig. 1). When this muscle contracts, the cartilage apparently is elevated.

The *tensor* veli palatini muscle is attached to the anterior wall. This fan-shaped muscle pulls downward and is supposed to act as an abductor.

Tubal obstruction. Tubal obstruction seldom is complete, and in most cases it is only partial. We find various degrees of obstruction around the mouth of the tube (central location) and/or farther up the tube in a peripheral direction.[10] As a rule the peripheral changes are secondary and subside when we treat the pathology in and around the mouth of the tube. *Tubal adenoids* cling to the lips or cushion of the tube. In chronic inflammation, the cushion becomes flabby and redundant. The lymphoid layer often becomes hyperplastic, causing a situation that amounts to follicular pharyngitis, which, of course, may be circumscribed, i.e., confined to the tubal area.

Histology. The *torus tubarius*, the blunt medial end of the tubal cartilage, is covered by the *eustachian cushion* (fig. 11), a very thick mucous membrane that is made up of the following layers:

1. Pseudostratified ciliated columnar epithelium
2. Basement membrane
3. Tunica propria
   a. A lymphoid layer along the posterior aspect of the cushion (this tissue always lies close to the epithelium, i.e., it is superficial)
   b. A glandular layer
4. Perichondrium

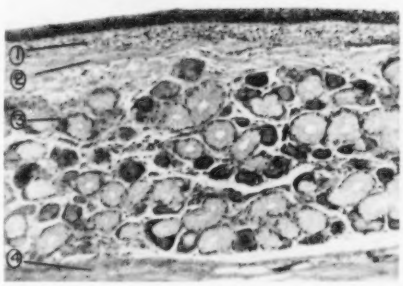

FIG. 17.—Mucous membrane lining the pharyngeal portion of the auditory tube (x300). (1) Subepithelial reticular layer and scattered infiltration of lymphocytes. (2) More fibrous layer of the tunica propria. (3) Glandular layer showing mixed mucous and serous glands. (4) Perichondrium.

The *auditory tube* is funnel-shaped, and as it funnels down, the lining membrane becomes thinner and all traces of lymphoid tissue practically disappear the moment we enter the orifice of the tube.

Along the roof of the tube, or "hook of Henle," the mucosa is thin, devoid of glands, and the lumen narrow. Throughout the remainder of the tube, the mucosa is well developed, endowed with an abundance of mixed mucous and serous glands, and lined with typical pseudostratified ciliated columnar epithelium. The tunica propria is not as areolar as that of the nasal sinuses. The submucosa contains glands and a variable deposit of *fat*.

CHRONIC EUSTACHIAN SALPINGITIS

The usual form of chronic salpingitis is a catarrhal inflammation. The salient features of the pathology are (1) hyperemia, (2) edema, (3) enlarged and hyperactive mucous glands, and (4) hypersecretion of mucus. Each of these changes plays an appreciable part in obstruction of the tube, particularly the mucus. In this respect the pathology is very similar to rhinitis, e.g., the swollen mouth of the tube becomes a mere slit and assumes the red color of infection or the pale, puffy one of allergy.

The epithelial changes are exactly like those seen in sinusitis.[35] We may find ciliary insufficiency, desquamation, hyperplasia, and goblet cell transformation. The basement membrane thickens slightly, along with some degree of thickening of the underlying tunica propria. Small round cell infiltrations may occur here and there, and a great many cells migrate through the epithelium. They contribute the mucous corpuscles to the thick discharge that forms in catarrhal inflammation. During catheterization of the tube, we can, as often as not, hear the rales, pop or rattle of the mucous plugs that obstruct the tube.

Allergic Salpingitis

Chronic salpingitis, like chronic rhinitis, frequently is due to allergy. The eustachian cushion, being more exposed to inhalent allergens, like the operculum of the middle turbinate, takes the brunt of the blow.

Chronic Follicular Salpingitis

Chronic follicular salpingitis may be defined as a histopathologic condition in which lymph follicles develop within the lumen of the auditory tube. It is rather rare and, in the imperfect state of our knowledge, cannot be diagnosed clinically.

Back in the days when the histology of lymphoid tissue was in its infancy, lymph follicles were called tonsillar tissue. The oft-mentioned Gerlach's tonsil (so-called tubal tonsil) dates back to the year 1875. It is not a tonsil in the true sense of the word, and even Teutleben[38] admitted that the slightest traces of lymphoid tissue may be totally lacking within the tube.

Moreover, the lymphoid tissue that the books mention is nothing more than a sprinkling of lymphocytes in the reticular layer of the tunica propria (fig. 17). We might perhaps speak of it as a small round cell infiltration.

The term "small round cell infiltration" is applied to cells resembling lymphocytes and is a noncommittal one, used when pathologists are unable to specify just which cells are present, i.e., lymphocytes, polyblasts, or plasma cells. The term applies to the small size of the cell and round shape of the nucleus. Small round cell infiltrations are typical of chronic inflammation almost anywhere in the body, and may be focal or diffuse.

To be capable of giving rise to tubal obstruction, lymph follicles must reach a diameter of about 0.5 mm. and be large enough to be seen with the naked eye. A solitary follicle is not a tonsil and, in the nose and sinuses, may arise as a consequence of a mixture of perennial allergy and infection.[35] We see them now and then in nasal polypi and also in aural polypi, where they apparently develop in the reticular tissue because of the inflammation.

A similar process takes place in the oropharynx. In this location, we call it chronic follicular pharyngitis.

The meager literature dealing with the histology of the tube does not offer enough statistical data to permit an accurate estimate of the actual incidence of follicular salpingitis. The necessary histologic material is difficult to obtain, and no one has an adequate collection of it at the present time.

The nodule shown in figure 16 lies near the isthmus of the tube and comes from a case of chronic suppurative otitis media.

In 200 histologic specimens of otitis media which I studied at the Johns Hopkins Otological Research Laboratory,[33] I found 3 tubes with nodules in them. The available material, unfortunately, was limited to the upper third of the tube. In 12 full-length sections of eustachian tubes (collected by E. P. Fowler, Jr.) which I examined while assigned to duty at the Army Institute of Pathology, one specimen had a few lymph follicles in various parts of the pharyngeal portion of the tube.

Wolff[43] stated that in a routine examination of 47 tubes, she found only one so-called tubal tonsil, and this specimen came from a child who had otitis media.

CHRONIC FOLLICULAR PHARYNGITIS

The lining of the mesopharynx (oropharynx) is made up of (1) stratified squamous epithelium, (2) reticular tissue, and (3) fibrous tissue. The glands of the posterior wall of the throat are wholly mucous; they lie in the fibrous tissue and sometimes penetrate the muscular submucosa. The term "reticular tissue" is another way of saying "microscopic lymphoid tissue," because one can give rise to the other. This type of tissue surrounds the excretory ducts, and we might assume that the constant presence of oropharyngeal infection demands it as a defense mechanism.

In some individuals, however, and especially those with recurrent attacks of pharyngitis and a tendency to allergy, the patches of lymphoid tissue increase in size and number and create a hyperplastic "granular or follicular pharyngitis." The rounded or oval nodules that characterize this condition vary in size from a pinhead to a large, flat patch the size of a dime.

Lateral bands arise in the pharyngeal recess, where we spoke of them as "lateral adenoids," and extend downward into the throat, forming a more or less continuous chain of lymph follicles behind the posterior pillars on either side of the pharynx. They become red and angry with acute inflammation and, between attacks, remain thick and hyperplastic. The epithelial surface often is rough and pitted with crypts. In children, they contain reaction centers; in adults, they consist mainly of a diffuse lymphoid tissue deposit and not a little fibrosis.

Clinical comments. The hyperplastic follicles of children may be nibbled from the underlying submucosa with a ring punch. In using the punch, I nibble the corn off the cob and do not take deep bites for rather obvious reasons.

When patients complain of chronic sore throat (the rheumatic throat of our forefathers) and the throat becomes infected and reinfected, the granular patches apparently perpetuate the pathology and pain. On several occasions I injected Cortisone directly into them but could see no immediate effects.

It has been said that removal of tonsils and adenoids causes a compensatory hyperplasia of the lymphoid patches. What causes the lateral bands and hyperplastic patches that we see before tonsils and adenoids are removed?

The Allergy Problem

Allergy and hyperplasia go hand in hand, and the overgrowth of lateral adenoids and lateral bands in allergic children is anything but a compensatory hyperplasia. What useful purpose does it serve?

If we view allergy as "a great imitator" and believe that nasal polypi serve no useful purpose, we may be able to approach the problem of chronic pharyngitis from a new direction.

Unfortunately, the criteria that we use in the histologic diagnosis of allergic nasal polyps hardly apply to lymphoid tissue. As a matter of fact, the clinical appearance of allergic epipharyngitis—the edema, pallor, bogginess, and succulence of the mucous membranes in and around the pharyngeal recess—is far more distinctive than the microscopic picture of allergic tonsillar tissue. Eosinophils, our most valuable cytologic clue to allergy, seldom stand out in tonsillar tissues, and when they do appear they are so outnumbered by the other leukocytes that we hardly notice them. Can we assume from this that hypersensitive lymphoid tissues react differently? If so, what bearing may this have on the histopathology of rheumatic nodules and collagen diseases, which is another way of saying "focal infection"?

TONSILLITIS

Nearly all of the inflammatory processes that characterize a tonsillitis begin in the crypts, or lacunae, as they sometimes are called. When these tortuous tubes become blocked either by compression of their walls or by cicatricial stenosis, how does the exudate and debris that accumulate in them escape? The epithelium is unciliated.

EPITHELIUM

The stratified squamous epithelium covering the surface of tonsils is fairly thick, and like epidermis it sheds freely. Upon entering the crypts, however, it often is reduced to one or two layers of cells. The basement membrane is almost nonexistent in this location, and in some places, the epithelial cells (large vesicular nuclei) and lym-

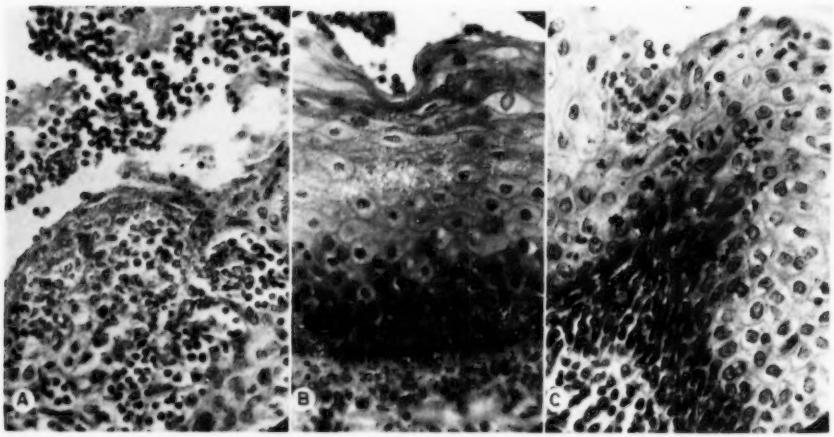

FIG. 18.—Faucial tonsils, stratified squamous epithelium (x600). (A) Lacunar epithelium. (B) Surface epithelium. (C) Emigrating polymorphonuclear leukocytes, purulent tonsillitis.

phocytes (intense dark-staining nuclei) create a line of scrimmage called lymphoepithelium by some authors and reticulation by others.

CRYPTS

There are ten to twenty crypts, some being tortuous, slender tubes, and others branched ones (fig. 19). Embryologically, they begin as epithelial buds that sink into the mesenchyme.[42] The lymphoblasts spring up around the buds, forming, at first, lymphoid tissue of the diffuse type, then primary nodules, and finally a single or double row of secondary follicles.

Bacteria enter the crypts shortly after birth and the battle is on. With bacteria in front of it and lymphocytes behind it, the epithelium turns this way and that to protect itself. It may resort to defense in depth and become thick (fig. 20).

Chronic tonsillitis *is* chronic because the attacks are recurrent.

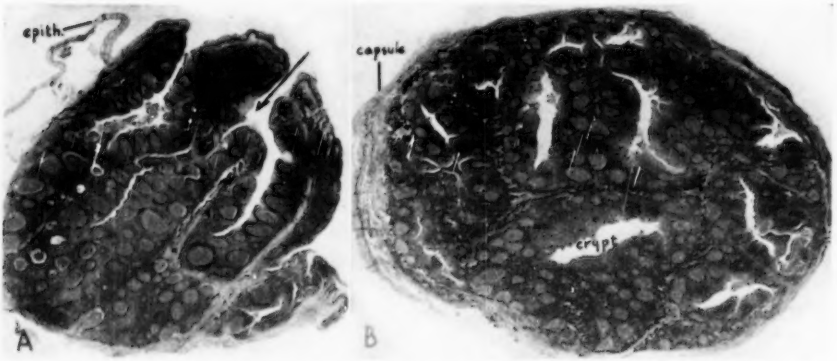

FIG. 19.—Faucial tonsils. (A) Sectioned from epithelium to capsule to show the depth of the crypts. (B) Cross section of the crypts show their tubular character. (x5).

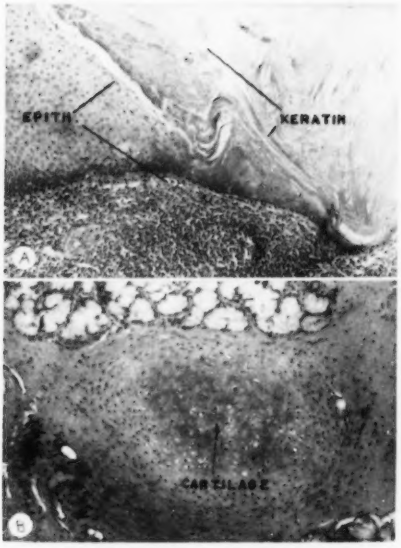

FIG. 20—(A) Hyperkeratosis of the lacunar epithelium in chronic tonsillitis. (B) Metaplastic cartilage in the capsule of a child with chronic infected tonsils.

Sometimes we find *fungi* (Leptothrix and Actinomyces) in the crypts, and when we do, we see much epithelial desquamation, as in otomycosis. Normally, the debris in crypts consists mainly of large numbers of lymphocytes. The fungi, it is said, are merely saprophytes. Can we become allergic to them?

The *bacteriology* of tonsillitis has been studied by a great many investigators interested in focal infection. Streptococcus hemolyticus seems to be the chief offender and certain strains enter into the etiology of rheumatic fever and other disturbances of a hypersensitive nature. Small protrusions of epithelium may be seen in the crypts and often contain blood capillaries, which is remarkable because most epithelium is avascular. Do these blood vessels permit bacteria to enter the blood stream?

TONSILLAR TISSUE

The lymphoid tissue of tonsils is exactly like that of the adenoids. The supporting stroma is made up of reticular tissue, collagen fibers, elastic fibers, fibroblasts, and intercellular substances. As has been stated, reticular tissue produces lymphocytes and forms a delicate basket around the follicles.

The capsule is well developed in the peritonsillar fossa, but much reduced over the mesial aspect (fig. 19). The underlying reticular tissue being soft and friable, we apply our forceps to the capsule during a tonsillectomy.

In children, the trabeculae are rather thin and made up of loose collagen fibers. With advancing age, the stroma becomes more collagenous, a process that is intensified by suppuration, e.g., fibrotic tonsillitis.

In *pyogenic tonsillitis*, the offending bacteria appear in the liquid pus that can be expressed from the crypts, but we have no way of seeing viruses, even though they frequently cause tonsillitis.

Acute Tonsillitis

Acute tonsillitis is characterized by much pain, reddening, swelling, and softening of the entire tonsil. Under the microscope we find gaps in the epithelium, areas of ulceration, polymorphonuclear leukocytes, plasma cells, large and small lymphocytes, macrophages, desquamated epithelium, pus and bacteria in the crypts.

Acute lacunar (follicular) tonsillitis is characterized by whitish or yellowish material in the crypts. The pathology, however, is not confined to the crypts, and invariably involves the follicles or reaction centers, creating swelling, infiltration of polymorphonuclear leukocytes, and areas of necrosis. The macrophages phagocytose bacteria; the bacteria persist; and the reaction centers

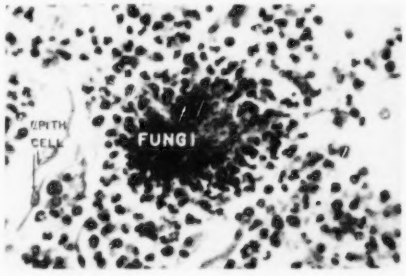

FIG. 21—Fungus colony surrounded by leukocytes and desquamated epithelial cells. This patient had recurrent attacks of tonsillitis with fever and anemia. The crypts contained pus cells and fungi. Figure 18 C is from the same case. Tonsillectomy cured this patient.

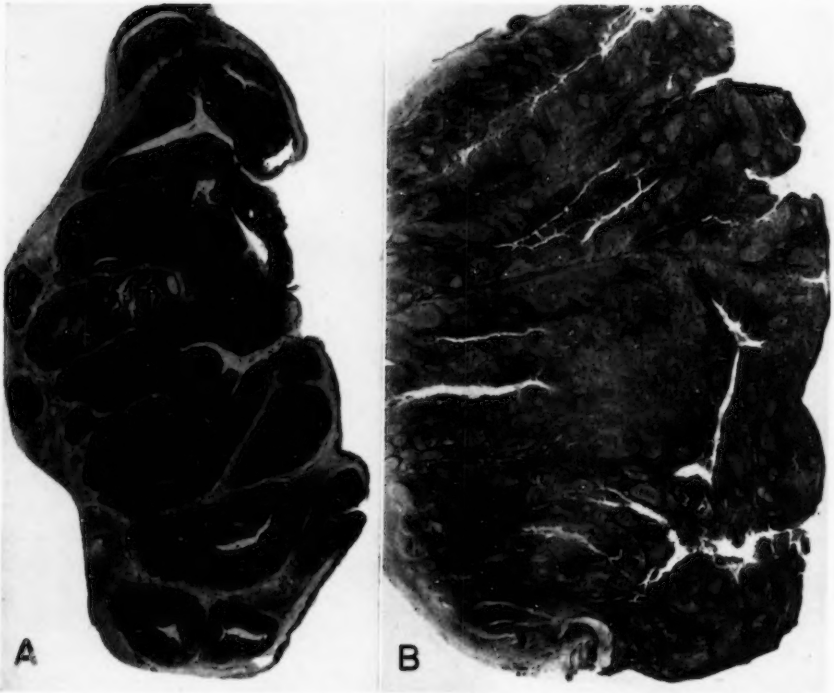

FIG. 22—Chronic hyperplastic tonsillitis. (A) Of an adult. Note the thickening of the trabeculae and capsule. (B) Of a child. Note the greater size of the tonsil and marked enlargement of the follicles (x5). The reaction centers are so large that they practically overflow and merge with one another.

break down into small abscesses. Histologically, as the intensity of the inflammation increases, the outlines of the follicles fade into the background of necrosis. The hyperemia and edema are intense and at times lead to hemorrhagic extravasation into the tissues.

Waldapfel[40] remarked:

> Acute tonsillitis is not a superficial infection; it involves the tonsil as a whole and is a deep-reaching phlegmonous infection with multiple abscess formation and destruction of large parts of the parenchyma. What appears as a "patch" is not a mere cast of exudate in a crypt, but quite as often an abscess perforating from inside the parenchyma to the surface of the tonsil.

The inflammatory hyperemia, leukocytic exudation and infiltration involves the capsule and connective tissue trabeculae. When the acute inflammation subsides, a certain amount of fibrosis ensues and small abscesses may remain behind, sometimes producing cysts in the depths of the tonsil.

Acute tonsillitis may lead to a number of complications (1) by direct tissue continuity, (2) by veins, e.g., jugular thrombophlebitis, and (3) by lymphatic vessels, e.g., cervical adenitis. When the acute infection persists, it may eventuate in (1) chronic lacunar tonsillitis or (2) suppurative tonsillitis.

Quinsy, or peritonsillar abscess, is an excellent example of *suppurative tonsillitis* (abscess formation). The infection may begin in the depths of a patho-

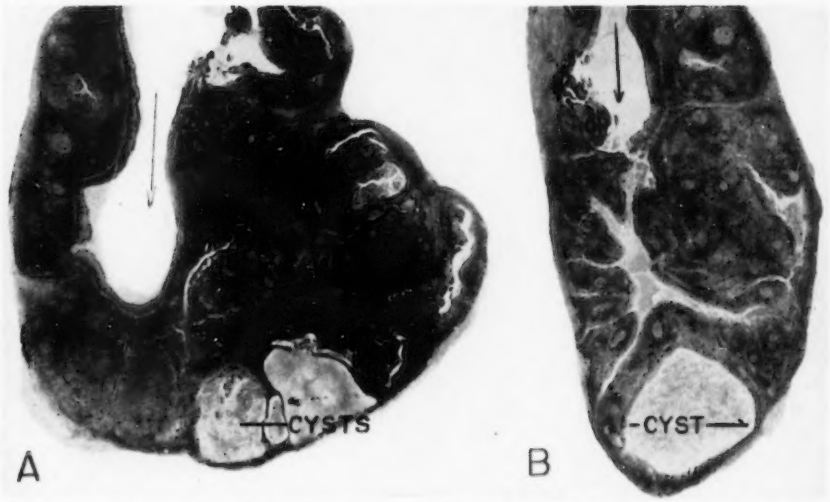

FIG. 23—Chronic atrophic tonsils. (A) From a patient with iritis. Note the cysts in the depths of the tonsil and diminished number of follicles. (B) From a patient with dermatitis (x5). A cyst containing crystals of cholesterin is present in this tonsil. The follicles are small and few in number. The size of a tonsil is no indication of the pathology in it.

logic crypt and dissect its way through the capsule into the surrounding areolar connective tissue. It may terminate in a number of fatal complications, erode the internal carotid artery, cause a deep neck abscess, jugular thrombophlebitis, or mediastinitis. With treatment, the suppuration subsides and leaves a trail of fibrosis and adhesions behind it in the tonsillar fossa. Recurrences are common, and this is one of the indications for surgery.

Chronic Tonsillitis

Some tonsils are buried, some semiburied, and others pedunculated, and so it goes with the pathology. One type merges into another without sharp lines of demarcation between the subtypes.

In *chronic hyperplastic tonsillitis* the tonsils are large and bulky. The increase in size is due largely to follicular hyperplasia. The epithelium of the crypts always is altered, and the crypts are filled with lymphocytes. Metaplastic inflammatory changes, the new formation of cartilage and bone, may occur in young children.

The salient feature, however, is the enlargement of the reaction centers, which sometimes become so large that they merge with one another (fig. 22). At the time of surgery, I have made touch smears of the mush that can be expressed from follicles, then have stained the slides with Hansel's stain in a search for eosinophils and other cells.

In *chronic lacunar tonsillitis*, the crypts contain malodorous plugs of thick, yellowish, cheesy pus or debris. Histologically, there sometimes is a foreign body reaction to the debris which may be mistaken for tuberculosis. Cysts are not unusual and may be situated in the depths of the tonsil, i.e., near the capsule (fig. 23). Gritty, calcareous material (calculus of the tonsil) may appear in the crypts.

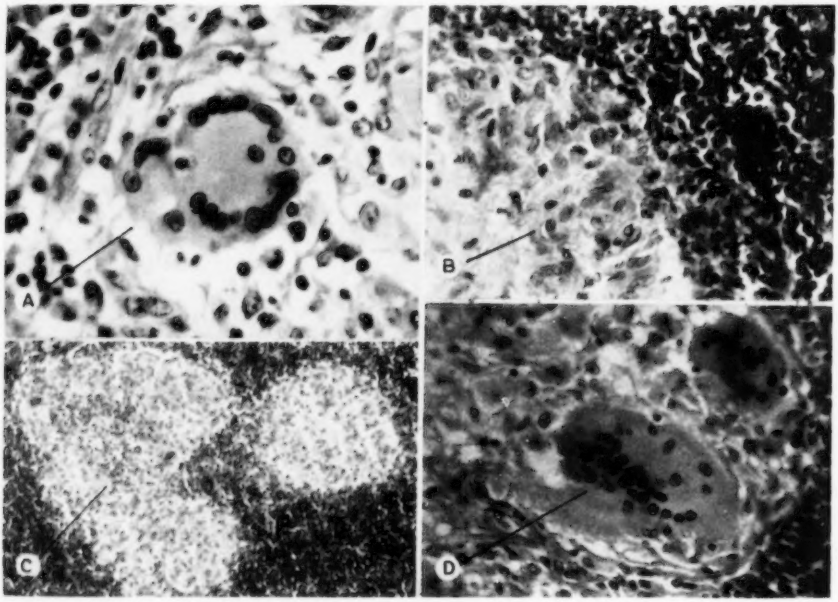

FIG. 24—Tuberculosis of tonsils. (A) Typical Langhans' giant cell (x1000). (B) Edge of tubercle showing epithelioid cells (x700). (C) Confluent tubercles (x70). At first glance they simulate follicles but are more irregular. (D) Epithelioid cells and giant cells (x700).

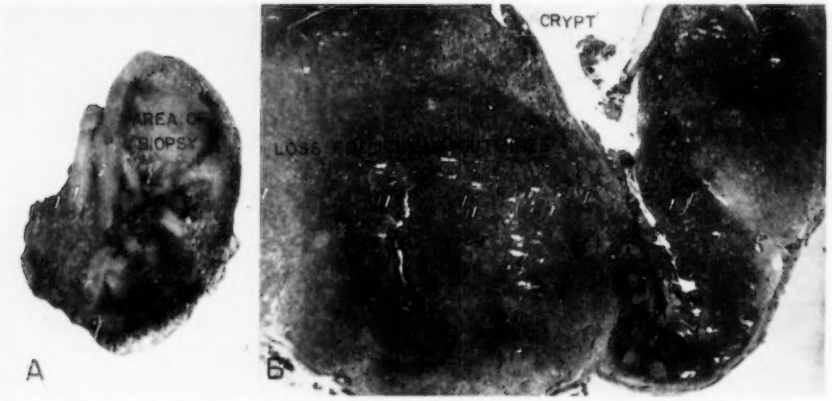

FIG. 25—Syphilis of the tonsils. (A) Gross specimen showing area of original ulceration and biopsy. The dark-field test was positive in this case. (B) Photomicrograph showing loss of outlines of the follicles.

In *hyperkeratosis tonsillaris*, horny plugs form in the crypts due to keratinization of the stratified squamous epithelium. The epithelium is thick and hyperkeratotic, and clinically, the spots look like raw tapioca. Smears will reveal fungus colonies (Leptothrix mycelia). The tonsils are of the atrophic type and the fibrosis may extend through the capsule into the musculature.

Sarcoid and tuberculosis sometimes may be found accidentally in surgical specimens. The tubercles are made up of epithelioid cells and giant cells.

Giant cells form by fusion of macrophages as well as by nuclear division without cell division. (1) The large multinucleated cell seen in tuberculous lesions is called the Langhans' giant cell. In this type, the nuclei are arranged in a horseshoe-shaped ring near the periphery of the cell. (2) In the foreign-body giant cell, they appear here and there in the cytoplasm. (3) The Touton giant cell is seen in xanthoma. (4) The lymphoid tissue giant cell (Dorothy Reed cell) is not as large as the others and may have one or two nuclei. It is associated with certain virus diseases and irradiated lymphoid tissue. (5) The osteoclast giant cell stems from osteoblasts and contains several nuclei in the middle of the cell. This giant cell is seen in tumors of bone and in epulis. (6) Malignant tumor giant cells are seen in sarcoma and anaplastic carcinoma and are very irregular in size and shape. They have multiple nuclei that are atypical. These cells actually are epithelial cells or sarcomatous cancer cells that become very large and assume giant shapes.

Tonsil Tags

Tonsil tags are of two types: (1) remnants of the original tonsil and (2) small, flat regrowths. The pathology of tonsil tags includes ulceration of the lacunar epithelium, keratinization, pus in the crypts, and fibrosis. The crypts may be dilated or distended with debris.[12] Boies[2] found tonsil tags in about 31 per cent of individuals; regrowths increased the incidence to about 50 per cent. Lymphoid follicles may occur in the plica triangularis as well as in the supratonsillar and retrotonsillar plica. Although these ingrowths are erroneously called tonsil remnants, they seldom are a source of disease.

The glands in the region of the tonsils are of the mucous type, and we often encounter a salivary gland in the supratonsillar region that, to the unaided eye, looks like a bit of tonsillar tissue. The early anatomists had the same trouble that we sometimes do trying to decide which is which.

Adenoid tags are more common than tonsil tags and for a number of reasons, the chief one being the poor exposure and anatomic difficulties that we must contend with in the nasopharynx. Another reason is the "out of sight, out of mind" attitude of those who minimize the special training and skill required in performing adenoid surgery. All adenotomes have limitations—to perform an adenoidectomy one must use the Love retractor, Yankauer speculum, and proper punches. Even so, it is almost impossible to reach the uppermost parts of the vault, and it is probably just as well, owing to the pituitary tissue in this area. The fossa of Rosenmüller and its adhesions are best handled with finger dissection, in children as well as adults. Adenoids tucked away in the pharyngeal recess frequently extend over the torus to the mouth of the tube and occasionally interfere with its motility. The tubal adenoids should be treated with the radium applicator. Good adenoid surgery is difficult to perform and conscientious surgeons do not hurry it or minimize its importance.

"SAY, AH!"

Although the tonsil problem can be summed up in two words—"Say, ah!," the knowledge that makes us specialists and not general practitioners hardly can be reduced to a few words about remedies. Like other problems, we approach tonsillitis from many directions. For example, we study and review the pathology in our surgical specimens (they are better than the illustrations

in books, which, after all, cannot show every detail), and the time we spend in this direction is well spent because the problem is a big one.

A million or more tonsillectomies, it has been said, are performed in the United States every year. If a single tonsil weighs three grams, and an adenoid about two grams, how many pounds, or even tons, of lymphoid tissue will be removed from men, women and children during our lifetime?

If Otolaryngology, the Cinderella of the specialties, is to endure the taunt of her sisters, let us face some questions.

Who is responsible for the rumor that removal of tonsils and adenoids will cause hay fever and asthma?

What is the difference between bacterial allergy and focal infection?

If tubal adenoids are located around the mouth of the tube, and in 80 per cent of the cases this is where we see the pathology clinically and histologically, who is in a position to pin-point the irradiation?

In the imperfect state of our knowledge, who advises us to irradiate the head from ear to ear? Is roentgen therapy that good?

Does the contention that one method of treatment is superior to another collapse when we look at pathology and not the apparatus at hand?

When Crowe and Burnam[6] demonstrated the effectiveness of pin-point irradiation, they accomplished two things: (1) they revived interest in the nasopharynx, and (2) they developed a nasopharyngeal radium applicator that strikes directly at the pathology (fig. 26).

When we get down to cases, every patient and his anatomic, allergic, endocrine and constitutional background is an individual problem. There is no cure-all for overlapping diseases, even in this era of antibiotics, antihistaminics, ACTH and radioactive substances.

Clinical experience has established the fact that lymphoid tissue is subject to rapid and intense inflammatory reactions. The acute reactions can be controlled with antibiotics, but what about the degenerative and obstructive lesions? Cortisone may control collagen diseases temporarily, but what about the foci of infection that sometimes, but not always, perpetuate or aggravate a fair number of these diseases?

As Arthur Proetz, in a provocative editorial, "Forty Questions in Otolaryngology," put it, "A fool can pose questions wise men cannot answer."[29]

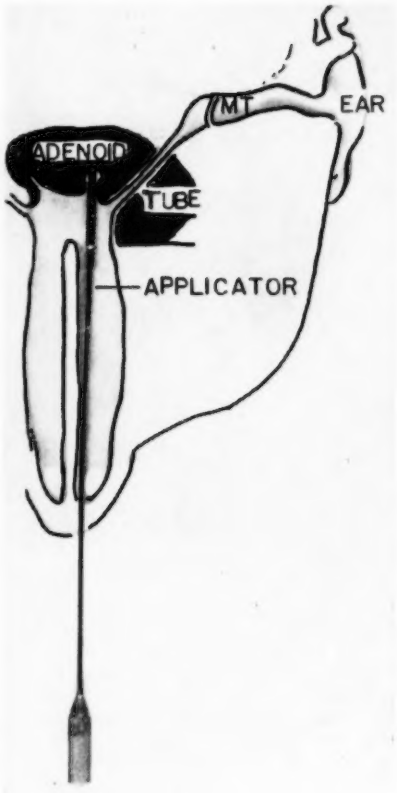

FIG. 26.—The nasopharyngeal applicator is shown in the pharyngeal area.

SUMMARY AND CONCLUSIONS

1. Knowledge of the pathology of lymphoid tissues, a subject that is fundamental to the practice of otolaryngology, is increasing day by day, and we are able to view it in a new light since the advent of ACTH and Cortisone.

2. The pharyngeal recess is filled with adenoid tissue which frequently extends to the posterior lips of the auditory tubes. Epipharyngitis is more common than is generally supposed, and we can recognize three types of inflammation in the nasopharynx: (1) allergy,

(2) infection, and (3) mixtures of allergy and infection.

3. There is little adenoid tissue within the auditory tube itself. The bulk of the infection in most cases of chronic eustachian salpingitis can be found in and around the mouth of the tube, particularly in the pharyngeal recess.

4. Adenoids occur in adults as well as children. When they cause symptoms and signs of disease, they should be removed surgically. There is no substitute for a conscientious adenoidectomy. Irradiation therapy is merely an auxiliary aid, a refinement that requires the expert eye and hand of an otolaryngologist. Small inaccessible adenoids clinging to the eustachian cushion or buried in the pharyngeal recess can be reached with the nasopharyngeal radium applicator.

5. To uncover this condition, the movements of the posterior lip of the tube should be examined routinely with an electric nasopharyngoscope.

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DISCUSSION

HENRY L. WILLIAMS, M.D., Rochester, Minn.: At first glance, Dr. Semenov's paper might seem trite, but I think that he has demonstrated with his presentation that there is a great deal for all of us to learn, or to relearn, in regard to this subject. I think some of us have departed from medical school so long ago that we may have forgotten the basis for diagnosis and treatment which our instructors tried to beat into us. Certainly it is evident that some men do not show much effect of early training. Instead of basing therapy and diagnosis on pathology, they have followed will-o'-the-wisps presented by people occasionally with an ax to grind but more frequently merely misguided.

It is high time that now, under the auspices of our President, who is so conversant with fundamentals, we should be again introduced to the pathologic basis for diagnosis and therapy. Dr. Semenov has demonstrated some points in therapy which have been neglected. It is quite obvious from his presentation that we cannot expect radium therapy in the nasopharynx to affect any but the nearest part of the eustachian tube. Radium therapy when it produces an effect produces it by destroying the lymphoid tissue in the epipharyngeal recess.

Dr. Semenov has also demonstrated the futility of expecting x-ray therapy to accomplish a great deal in syndromes of the eustachian tube.

I think it is time that radium treatment of the nasopharynx be rescued from the general practitioner and returned to the otolaryngologist. We have been remiss, but our interest has been reawakened in some of the basic sciences, and we should now be more capable of handling these matters.

Owing to confusion of the hypothesis of focal infection with the hypothesis of selective localization of bacteria, many medical men have hesitated to advise the removal of the tonsils or foci in conditions in which that practice should be followed by a favorable outcome. The possibility of relationship of focus to secondary pathologic area by the antigen-antibody reaction has been demonstrated by Dr. Semenov in these slides.

I personally feel that our President has done a great deal for us in insisting on this largely pathologic program. I for one feel that I need it, have profited a great deal by it and have relearned lessons which I should not have forgotten.

I want especially to thank Dr. Semenov for his splendid presentation of this subject.

BEN H. SENTURIA, M.D., St. Louis, Mo.: Dr. Semenov has presented a very interesting and provocative discussion of the problems relating to lymphoid tissue in and around the nasopharynx. I would like to comment and possibly elaborate on a few problems upon which he has touched.

Only a few in our specialty have written about the problem of direct visualization of the nasopharynx. A review of the literature disclosed the views and measurements of von Gyergyai. Shortly thereafter Yankauer presented the speculum which bears his name. Some years later Kelly described his direct vision adenotome. Subsequently Love and Beck described instruments and technics for retracting the soft palate.

In our hands these instruments do not permit good visualization of the roof of the nasopharynx or the pharyngeal recesses. One may assume some limitation or inherent defect in these instruments from the fact that they are not widely used. Recently I have attempted to overcome some of these objections, and we now have available a nasopharyngeal speculum which, in children particularly, allows for good visualization of the inaccessible areas and permits easy use of a biting forceps and suction in the nasopharynx.

Dr. Semenov comments on the difficulty in differentiating the allergic hyperplasia from the infected follicular hyperplasia. My associates and I have established a routine in the office which simplifies the problem in a high percentage of the cases. It is our practice in these cases to obtain a careful allergic history and a thorough examination which includes nasal smear, blood smear, and roentgenograms. It is gratifying to be able to sit down with the patient on his second visit and point to the positive history of allergy, the pale nasal mucous membrane, the increased blood and nasal eosinophils and possibly a negative roentgenogram, and explain that these findings substantiate the impression of an allergic follicular hyperplasia. On the other hand, in a case of infected follicular hyperplasia we can frequently point to the clouded antrum on the roentgenogram, the myriads of neutrophils on nasal smear, the absence of eosinophils in the blood smear, and the negative family history or past history of allergy.

I would like to refer briefly to the problem of radium and roentgen therapy to the nasopharynx. If there is present widely distributed adenoid tissue, we prefer re-adenoidectomy. If surgery is refused, at the present time we advise roentgen therapy and base this advice on the very experimental evidence Dr. Semenov presents. On the other hand, if the lymphoid tissue is restricted to a small area in and around the nasopharyngeal ostium of the eustachian tube and this is interfering with satisfactory ventilation of the middle ear, we localize our therapy by the use of radium applicators.

Allow me to discuss with you one more thought prompted by Dr. Semenov's presentation. Many of us have been asking ourselves, how much lymphoid tissue shall I remove? Is it possible to be too radical in the removal of nasopharyngeal lymphoid tissue? I do not go as far as I believe Robison does, but we do clean out any adenoid tissue which appears to interfere with good nasal or aural ventilation. I remove as much lymphoid tissue as necessary from the roof of the nasopharynx to clear the posterior nares. Any lymphoid masses in the pharyngeal recess which appear to interfere with good function of the eustachian tubes are bitten away. We do not hesitate to nibble away at the salpingonasal and salpingopalatine folds which appear to impede good nasal ventilation. We have seen no bad results from stripping away, with a scissors, the lymphoid masses on the salpingopharyngeal fold.

I need not point out to this audience that good judgment is required in doing this kind of adenoid surgery. One must be careful to avoid adhesions between the tube and adjacent membranes. Certainly injudicious use of the biting forceps or scissors will cause intractable bleeding. On the other hand, I cannot subscribe to the recommendation that one must observe caution in the removal of a few millimeters of questionably active aberrant pituitary tissue in the nasopharynx and chance the leaving of adenoid tissue which may act as a focus for spread of infection to the nose and the ear.

I'm sure we are all grateful to Dr. Semenov for such a stimulating and thought-provoking paper.

MEGAESOPHAGUS (ESOPHAGEAL DYSTONIA) AND ESOPHAGITIS

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MINNEAPOLIS, MINN.

BY INVITATION

ESOPHAGITIS

OBSERVATIONS from this clinic relative to the origins of so-called idiopathic strictures of the esophagus have been documented elsewhere.[15] On the first Sunday in September, 1939, I did a resection for a middle aged man with a bleeding duodenal ulcer, because of massive hemorrhage (fig. 1a). Over a period extending back to 1935, he had been dilated more than 100 times in the Out-Patient Clinic. After the resection, I told him he would have to continue the dilatations. After two dilatations he refused further therapy, insisting that he no longer needed dilatation. He remains well today (fig. 1b). There have been no additional dilatations in the intervening thirteen years.

Three years went by and a similar sequence of events was noted in a second patient who had suffered from a peptic ulcer of the duodenum for years. He was just getting to the place where he was in need of dilatations. A gastrojejunostomy had been done earlier, and when seen in 1942, he had a stomal ulcer as well as a constricting esophagitis. Again, gastric resection was followed by complete cessation of difficulty in swallowing. This second case made me see the light. I knew now there was some relationship between the reflux of acid-peptic juice and the occurrence of an esophageal stricture.

Two additional years went by before I saw another case. The long war years with a great shortage of man power in the Clinic had made it virtually mandatory to abandon all but military linked surgical research in the laboratory. This third patient had no duodenal ulcer, as did the two earlier patients. Moreover, she had a corkscrew-like stricture approximately 10 cm. above the cardia (fig. 2a). She, too, had been dilated more than 100 times. She accepted my suggestion of gastric resection in the light of the two other successes, even though she knew, too, that no one else on the Staff agreed with the suggestion. She, too, demonstrated dramatic improvement (fig. 2b). Two dilatations of the esophagus were necessary in the early convalescence. None have been done since. It is to be noted that films made in 1951, seven years after gastric resection, show a fairly normal esophagus (fig. 2c). The corkscrew-like stricture has disappeared.

The most striking case of all concerns a man who carried a gastrostomy, made eight years previously because he could not swallow his saliva. This man had a

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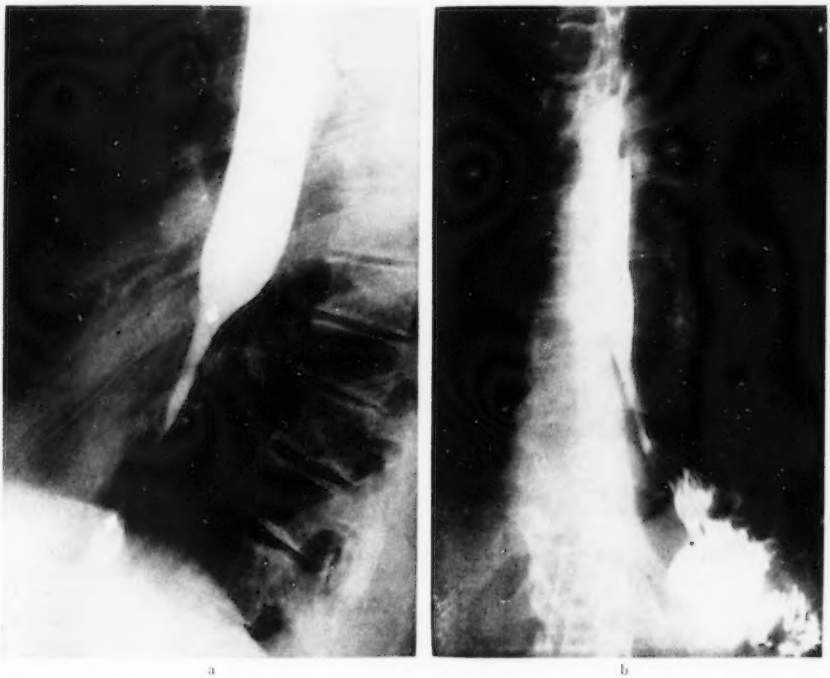

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FIG. 1—Cure of stenosing esophagitis after gastric resection performed for massive hemorrhage from a duodenal ulcer. a. Esophagogram made April 1, 1936; there is slight dilatation of the esophagus above the stricture. This patient had undergone more than 100 dilatations prior to the gastric resection. b. Esophagogram made April 3, 1948, almost nine years after gastric resection, which was performed September 2, 1939, for a bleeding duodenal ulcer. The patient is symptomatically well, and there is no demonstrable esophageal narrowing. Following two esophageal dilatations, performed following the gastric resection, the patient refused further dilatations. The esophagus now admits a no. 46 F. bougie without difficulty.

known duodenal ulcer; moreover, the films showed the presence of a crater in the duodenum at the time of his admission to the University Hospital. The length of the stricture can be seen in figure 3. It was possible to pass a no. 5 urethral catheter into the stomach and to bring it out through the gastrostomy wound. By replacing it with a silk thread, the esophagus was gradually dilated to take a no. 19 F. bougie. Then gastric resection was done. The patient improved slowly. Presently, a no. 23 F. bougie could be passed. At lengthening intervals, the patient was dilated over a period of six months until a no. 37 F. bougie could be passed. Since then no dilatations have been carried out. Some months ago, four years following his operation, the patient returned again for observation. A no. 42 F. bougie was passed without difficulty. The patient eats celery, carrots, hard rolls and steak without difficulty. In fact, he considers himself completely well.

In all these cases, a three-quarter gastric resection was carried out on the Billroth II plan of operation. In this procedure, adequate provision is made

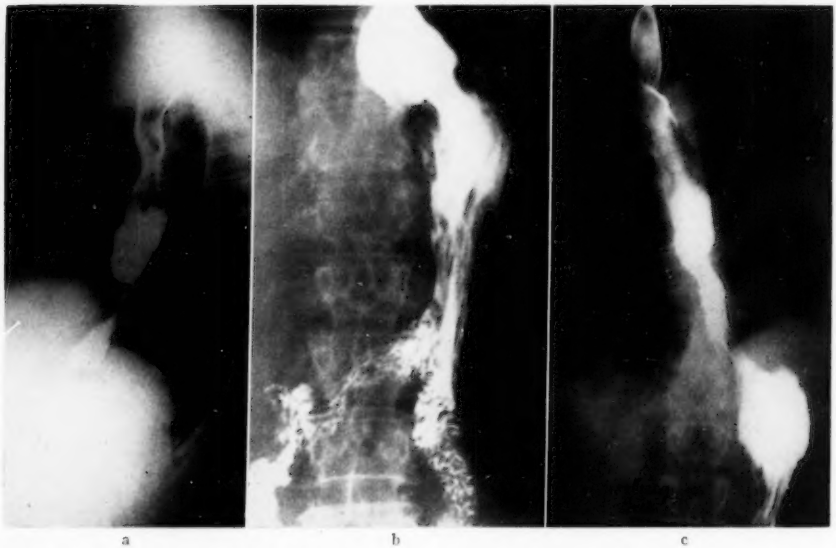

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FIG. 2.—Corkscrew stricture of the distal third of the esophagus of many years standing cured by gastric resection. This patient did not have a duodenal ulcer. a. Film made in April 1944, a few months prior to operation. b. Film made shortly after gastric resection, which operation was done August 14, 1944. c. Film made in October, 1951, seven years after gastric resection. The esophagus looks quite normal. Whereas, more than 100 dilatations of the esophagus had been done prior to operation, only two were done in the early convalescent phase; none since.

to ensure satisfactory gastric drainage, and the capacity of the stomach to secrete acid is reduced considerably by the extent of the excision of acid-secreting area in the stomach. In more recent years, idiopathic esophageal strictures have been treated successfully by segmental gastric resection as well as by tubular gastric resection.[14] (See footnote.) In these latter procedures, it is important, obviously, to add an adequate pylorotomy of the Heineke-Mikulicz variety to afford ready drainage from the stomach.

Now I was not astute enough to prevision these dramatic occurrences. In fact, I was only a passive observer in the first two patients, who had compelling indications for urgent surgery for peptic ulcer. In other words, it was a serendipitous adventure, to use a phrase coined by Horace Walpole, a gratuitous and unexpected result from an operative procedure invoked to relieve the sequelae of peptic ulcer. In the last two instances, however, the esophageal stricture was the compelling indication for operation.

It would be fair, I believe, on the basis of these observations to suggest that idiopathic stricture of the esophagus is, in all likelihood, acid-peptic stricture of the esophagus, occasioned by the reflux of acid-peptic juice into the esophagus from the stomach.

When the long war came to an end, it was possible to submit the thesis of a

Since this paper was prepared, two somewhat more complete references have been made to the subject of tubular gastric resection: (1) "Evolution and Evaluation of an Acceptable Operation for Peptic Ulcer," which will appear in the September issue of the Review of Gastroenterology, 1953; and (2) "The Peptic Ulcer Problem," which will appear in Minnesota Medicine in the September issue, 1953.

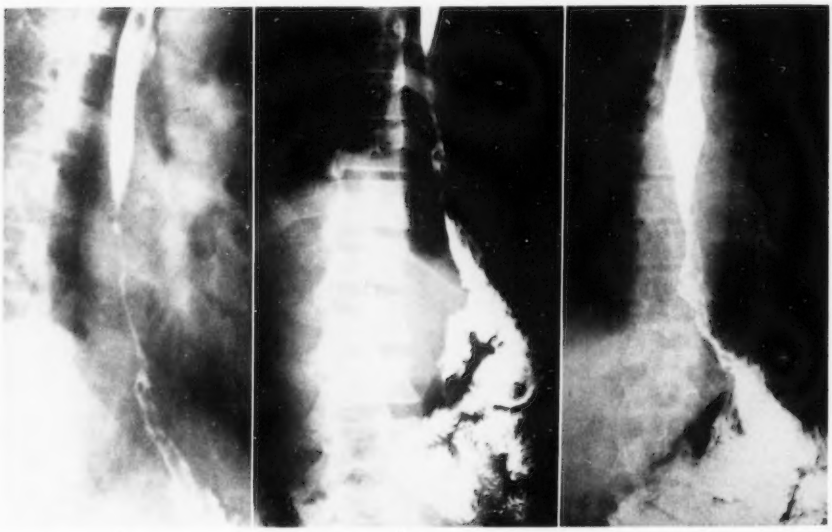

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FIG. 3.—This man carried a gastrostomy for eight years prior to operation because finally he could not swallow his saliva. a. Film made in June, 1947. There is a long narrow stricture involving the distal half of the esophagus. b. Film made shortly after gastric resection, March 19, 1948. c. Film made in September, 1951, approximately three and a half years after operation. Obviously, there has been remarkable improvement.

Prior to gastrostomy, this patient was dilated an "innumerable" number of times. Just prior to the operation, the stricture was dilated so that a duodenal tube could be pulled through the esophagus. Following operation, he was dilated at increasing intervals for a period of six months. There have been no dilatations during the past three years. The esophagus now admits readily a no. 42 F. bougie.

low threshold of susceptibility of the esophageal mucosa to injury by acid-peptic juice to experimental scrutiny.[11] It was done in the manner outlined in the next caption.

"SPONTANEOUS" PERFORATION OF THE ESOPHAGUS

On the basis of the observations cited above, the following experiment was done. In eight dogs, the pylorus was obstructed and the dogs were given 30 mg. of histamine base in beeswax intramuscularly each day, supported by intravenous therapy of fluid and electrolytes. In all eight dogs, at the end of four days, severe esophagitis was present. The stomach remained quite normal. In four of the eight dogs there was a linear perforated ulcer in the esophagus. I stress the word *linear* because the apostles of the hydrostatic theory of spontaneous perforation suggest that one would expect a *round* ulcer if these were acid-peptic ulcers. However, the observed ulcers were linear or elliptical in shape (figs. 4 and 5).

This very susceptibility, therefore, of the esophageal mucosa to injury by acid-peptic juice lends credence to the belief that the reflux of acid into the esophagus is probably the origin of idiopathic esophageal strictures observed in man.

If one takes juice from the isolated gastric pouch of a dog and drips it into the esophagus of another dog, the stomach remains quite normal but the esophagus is easily damaged. Linear

FIG. 4—Perforation of the dog's esophagus following obstruction of the pylorus. Histamine in beeswax (30 mg.) was given daily intramuscularly over a period of four days, the dog being maintained by the intravenous administration of fluids and electrolytes meanwhile. When the dog was sacrificed four days later, the stomach was normal; there was a greyish swollen appearance of the entire esophageal mucosa. The linear perforation in the lower esophagus is readily discernible.

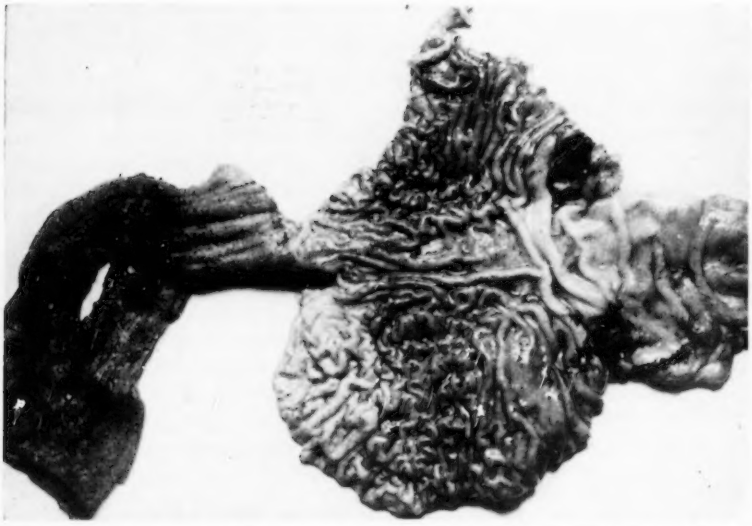

FIG. 5—Linear perforation of the esophagus following the dripping of gastric juice into the oral end of the esophagus. The upper fundic end of the stomach was tied over the outlet cannula, and an intraluminal pressure within the esophagus of 20 cm. of fluid was maintained.

The gastric juice employed in the dripping was obtained from the isolated gastric pouch of another dog. The pH of the gastric juice employed in the dripping was 1; when hydrochloric acid of the same pH is infused, severe esophagitis follows; however, perforation was not observed.

perforation was observed quite regularly; in fact, in the cat, perforation has been observed as early as twenty minutes following commencement of the dripping of gastric juice obtained from a dog's isolated gastric pouch.[4] And after two hours of dripping, perforation of the cat's esophagus was observed frequently. The dripping of hydrochloric acid alone with pepsin, though followed by evidences of injury to the esophagus, is not accompanied by perforation, attesting the validity of the time-honored custom of including the designation "peptic" in describing the origin of acid-peptic ulcer.

Another interesting experiment bearing on the ease of damage of the esophageal mucosa by acid-peptic juice is suggested in the following: when a small, penny-sized bit of gastric mucosa has been elevated with its blood supply intact and implanted into a small aperture made in the wall of the esophagus, perforation occurs quite regularly just opposite the pedicled gastric mucosal graft.[2] This occurrence is observed even after the remainder of the stomach has been excised, so sensitive is the esophageal mucosa to injury by acid-peptic juice.

Hemorrhagic erosions can be produced in the dog's esophagus by attaching that segment of the duodenum containing the bile and pancreatic juice to the wall of the esophagus (fig. 6). Moreover, either bile or pancreatic juice alone may produce such erosions, but the combined effect of the two juices in the segment is followed by more palpable evidences of injury to the mucosa of the esophagus. When the duodenal segment is anastomosed to the lateral wall of the esophagus after exclusion of the bile and pancreatic juice, no erosions are observed in the esophagus (fig. 7).

Such, in brief, has been the background of interest of my colleagues and

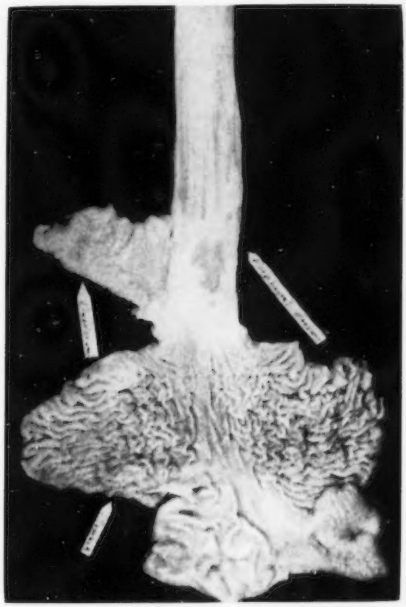

FIG. 6—Esophagitis provoked by affixing the bile and pancreatic juice containing segment of the duodenum to the lateral wall of the esophagus. Whereas, many such dogs developed anemia, perforation of the esophagus was not observed 3a.

myself in the susceptibility of the esophageal mucosa to injury by the digestive juice. Allison[1] has called attention to the circumstance that a paraesophageal hernia may predispose to injury of the mucosa of the esophagus in man, followed by stricture formation. In the patients reported herein, in whom gastric resection was followed by relief of an idiopathic esophageal stricture, no evidence of hiatal diaphragmatic hernia was observed at operation, suggesting that the reflux of gastric juice may occur in the absence of any demonstrable defect in the diaphragmatic sling surrounding the terminal esophagus. As long ago as 1926, Robins and Jankelson[10] demonstrated that the regurgitation of barium from the stomach into the esophagus could be observed on the fluoroscopic

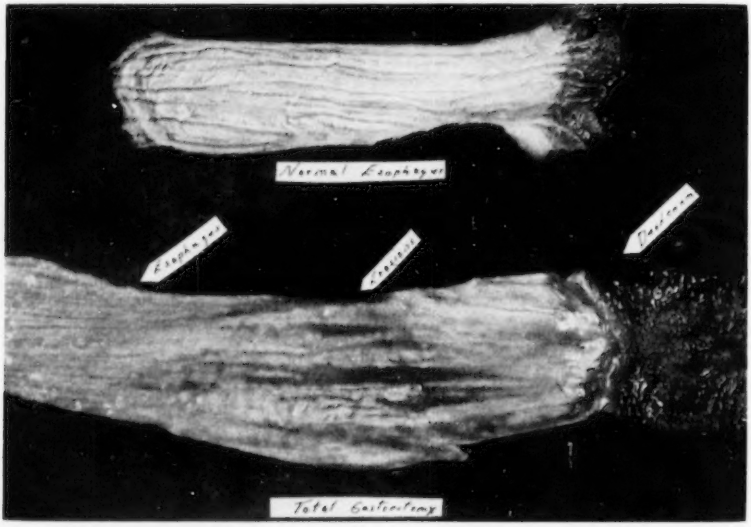

FIG. 7.—Esophagitis observed in a dog attending total gastrectomy, the esophagus being united to the duodenum end-to-end. A ready reflux of bile and pancreatic juice occurs into the esophagus. In man, the preferred method of restoring continuity after total gastrectomy is by the Roux Y principle of restoration.

screen in 4 per cent of patients, when the patient was placed prone on the x-ray table. These observations, obviously, should be extended.

ETIOLOGY OF MEGAESOPHAGUS

The origins of megaesophagus remain an enigma. There are essentially two components of the disease: (1) hypertonus of the terminal segment of the esophagus, the so-called pinch-cock effect of Chevalier Jackson, and (2) atony of the more proximal reaches of the esophagus.

The very presence of a dilated esophagus helps to establish the diagnosis. A tonic esophagus maintains its tone when obstructed and does not dilate. This circumstance is evident in the films of patients with esophagitis shown in figures 1 to 3. Unusual dilatation of the esophagus is synonymous with megaesophagus.

Atrophy or disappearance of the ganglion cells of Auerbach's plexus is noted on histologic examination in cases of megaesophagus, much in the same manner that these cells are absent also in Hirschsprung's megacolon.

Dilatation of the esophagus may be provoked in the dog by extensive vagotomy, an observation made by Wilhelm Rieder[9] approximately 20 years ago and confirmed by McManus and his associates[7] at Buffalo more recently. Moreover, McManus and his associates failed to note any disappearance of the parasympatnetic ganglion cells in Auerbach's plexus attending division of the vagus nerves.

TREATMENT OF MEGAESOPHAGUS

Having had no experience with instrumentation or endoscopic procedures on the esophagus, I can only say that the simplest treatment for the condition,

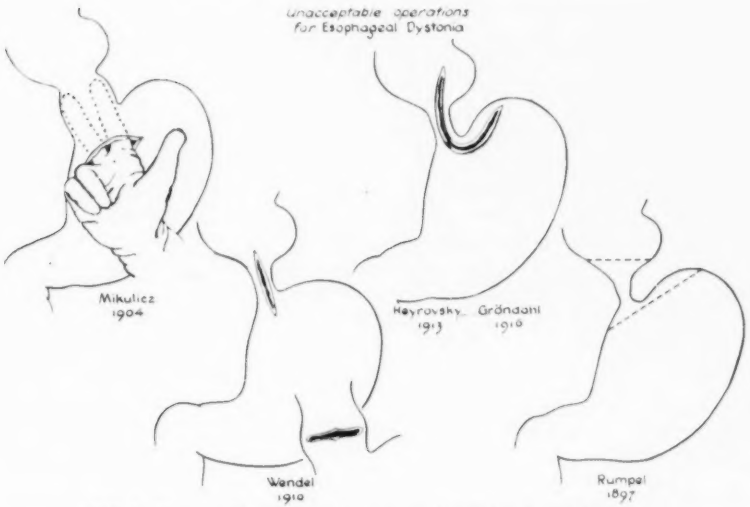

FIG. 8—Unacceptable operations for megaesophagus (esophageal dystonia).

when effective, is a bloodless myotomy, accomplished by hydrostatic dilatation. When successful, this procedure affords the patient, ordinarily, complete relief from the dysphagia. Usually, however, it is my impression that dilatation of the esophagus still persists. The unfortunate circumstance is that this procedure is not uniformly successful. It would appear that two attempts at dilatation will relieve 50 per cent of the patients. Olsen and his associates[8] at the Mayo Clinic report a somewhat higher incidence of success with hydrostatic dilatation. Sifers and Crile[12] noted evidences of clinical improvement in approximately 30 per cent of their patients submitted to hydrostatic dilatation. A group of observers[5] from the clinics of the University of Chicago indicate that they are not enthusiastic over hydrostatic dilatation for megaesophagus. Moreover, Sifers and Crile noted that chills and fever were a frequent complication of dilatation, a circumstance which suggests that an occult perforation may have occurred attending the dilatation.

OPERATIONS FOR MEGAESOPHAGUS

Unacceptable operations[13] for the relief of megaesophagus or esophageal dystonia are illustrated in figure 8. It was Mikulicz who attached the name cardiospasm to the condition in 1882. In 1904, he suggested transabdominal dilatation of the hypertonic segment of the esophagus through a gastrostomy opening. In the main, this has not been a very satisfactory method of dealing with the situation. The Wendel (1910) open cardioplasty is, apparently, followed quite regularly by evidences of esophagitis, the reason being that a complete linear slit of the esophageal wall followed by transverse closure of such a slit permits reflux of acid-peptic juice into the esophagus.

Heyrovsky (1913) made an esophagogastrostomy between the dilated esophagus and the fundic end of the stomach. Gröndahl (1916) made the esophagogastric anastomosis, employing the Finney pattern of plasty. This latter procedure, now known as the Heyrov-

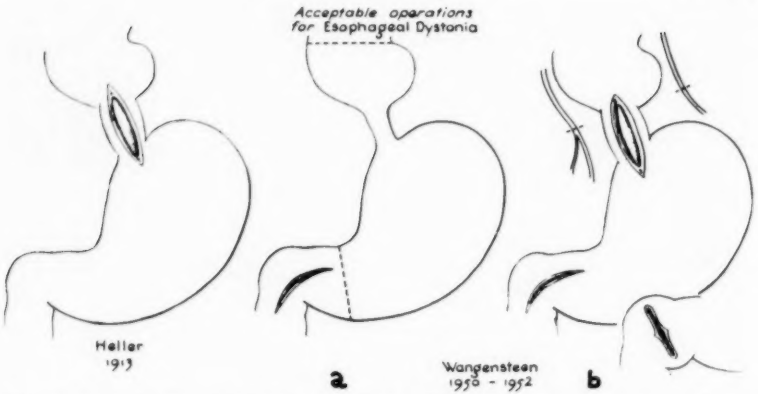

FIG. 9—Acceptable operations for megaesophagus (esophageal dystonia). The Heineke-Mikulicz pyloroplasty ensures ready emptying of the stomach, thus lessening the hazard of esophagitis.

sky-Gröndahl operation, is obviously not a good operation for esophageal dystonia in that it too obviously invites regurgitation of gastric juice into the esophagus.

Sympathectomy as an operative procedure directed at the relief of esophageal dystonia obviously has no place in the treatment of megaesophagus.

ACCEPTABLE OPERATIONS FOR ESOPHAGEAL DYSTONIA

My own personal experience with the Heller operation has not been great. Yet, from the literature, it is obvious that this operation, though in principle very similar to the Wendel procedure, is nevertheless a far more acceptable operation on the basis that it is not apparently followed by evidences of esophagitis (fig. 9). The testimony of Barrett and Franklin[3] of London on this score is rather impressive. Moreover, Kelly, Brackney and I[6] have noted in dogs that reflux of gastric juice with evidences of esophagitis follows quite regularly when the Wendel open cardioplasty is done, whereas esophagitis has been absent in dogs following the Heller extramucosal myotomy.

Use of the Bardex Balloon-Tipped Catheter in the Heller Operation

Owing to the circumstance that the Heller procedure in my hands has failed to reduce the size of the dilated esophagus, in the last patient upon whom I tried the procedure I employed a Bardex catheter with a concentrically arranged balloon (fig. 10). Following the

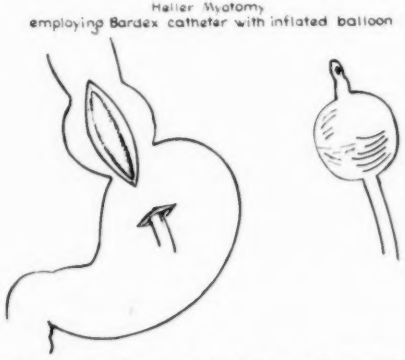

FIG. 10—Employment of the Bardex catheter with inflating balloon in the Heller extramucosal myotomy. A long incision is made with a knife. The catheter is introduced through a gastrostomy wound. The final rupture of any remaining muscle fibers in the mucosa is accomplished by repeated inflation of the balloon at various levels of the myotomy incision. Air is employed for the inflation because it is more labile than water. In other words, deflation of the balloon can be done more rapidly.

placement of a linear extramucosal slit in the esophagus and the upper portion of the stomach, cutting most of the muscle fibers, a Bardex catheter was introduced into the lower reaches of the esophagus through the gastrostomy wound and the unbroken residual circular muscle fibers were ruptured in this manner by repeated inflations of the balloon (fig. 11). Interestingly enough, this patient, within three weeks after the operation, presented an esophagus which was quite normal in outline, with return to normal size (fig. 12). In other words, it would appear that a more complete rupture of the fibers of the circular muscle fibers is achieved by the use of the balloon-tipped catheter, as contrasted with division of these muscle fibers with a knife. Moreover, at operation, the balloon was inflated at several areas in the lower esophagus so that the remaining esophageal mucosa finally appeared almost diaphanous, suggesting definitely that all the muscle fibers had been effectually ruptured. When bloodless myotomy is done by the hydrostatic method, one might reasonably infer that complete rupture of the muscle fibers would also be followed by return of the dilated esophagus to normal size. Inasmuch, however, as dilatation of the esophagus usually persists despite a satisfactory functional result when hydrostatic dilatation is successful, it would appear likely that rupture of the circular muscle fibers is ordinarily incomplete.

The case history of the patient, in whom the Bardex catheter to complete the extramucosal Heller myotomy was employed, follows.

FIG. 11.—Photograph at operation with the Bardex catheter in place. This excellent surgical exposure was obtained through a midline upper abdominal incision, completed by an extrapleural sternal split extending into the fourth interspace on the left side.

CASE REPORT

This 53-year-old white male entered the hospital for the first time on September 9, 1952, with the complaint of difficulty in swallowing food of a year's duration. At first the difficulty was mainly with meat, but the impediment to swallowing increased gradually to the point that during the last six months he had been able to eat only such soft foods as soups, milk, eggs, and cereal. Attempts at ingesting more solid foods resulted in their being regurgitated. There has been no pain, nor has there been hematemesis or melena.

During the past year, the patient has lost 60 pounds in weight. The previous past health has been good. There have been no symptoms of peptic ulcer, no previous surgery. On examination, the patient was thin, exhibiting evidences of recent weight loss. There were varicose veins of the lower extremities.

The hemoglobin was 15 gm., leukocyte count 8,750, urine negative. Gastric analysis indicated 28% free hydrochloric acid. The ordinary laboratory data were within normal range.

X-rays of stomach and esophagus were performed on September 10: There was slight dilatation of the esophagus with an area of narrowing at the distal end of the esophagus, fairly typical of the pinch-cock effect observed in patients with cardiospasm. The outline of the esophagus appears quite smooth. The appearance is not typical of a well-developed megaesophagus. No organic obstruction is found.

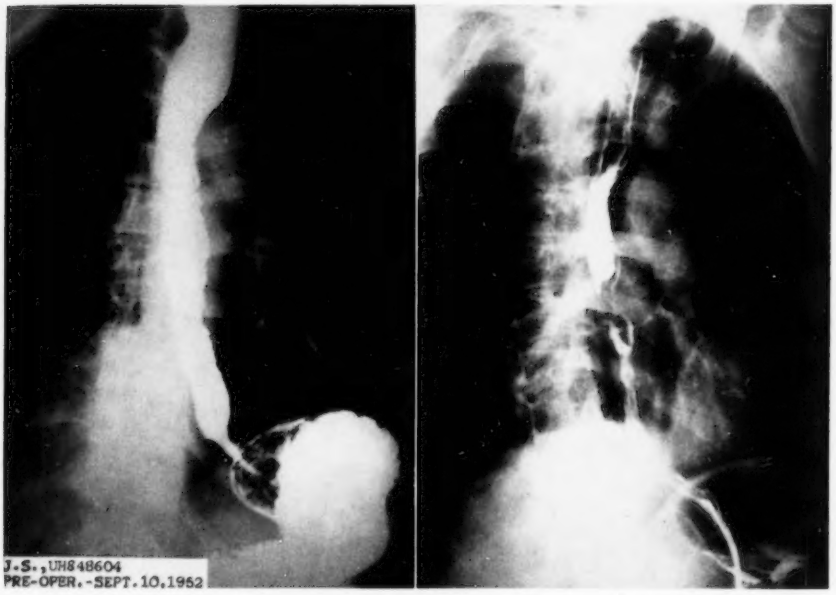

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FIG. 12—Esophageal dystonia of a year's duration in the case of Mr. J. S., aged 53. Attempts at hydrostatic dilatation had been unsuccessful. a, Before operation. There is only moderate dilatation of the esophagus. The hypertonus of the distal segement of the esophagus is quite evident. The patient lost 60 pounds during the year prior to operation. b, X-ray film made three weeks after the operation. The esophagus has already shrunk to normal size. Whether this circumstance is owing to more complete division of the muscle fibers or to evidences of some tonicity of the more proximal reaches of the esophagus as is shown in a, is difficult to say. There may be an element of both in the final answer.

Surgery, September 15: A midline upper abdominal incision with sternal splitting. A gastrostomy opening was made in the fundus. A finger placed through this opening revealed a suggestion of a narrowing of the cardia and the terminal end of the esophagus. A Heller extramucosal myotomy was performed, employing the Bardex catheter. The usual long extramucosal linear slit was made in the distal 80 cm. of the esophagus, including an additional length of 6 cm. of the cardiac portion of the esophagus. The Bardex catheter was employed to rupture the last circular muscle fibers. The balloon was placed successively at 3 levels and inflated until every visible persistent muscle fiber was ruptured. The mucosa finally appeared almost diaphanous. A vagotomy was done to diminish gastric secretion. A Heineke-Mikulicz pylorotomy was also done to ensure ready emptying of the stomach.

September 30: The patient is eating well; also feels well. No dysphagia.

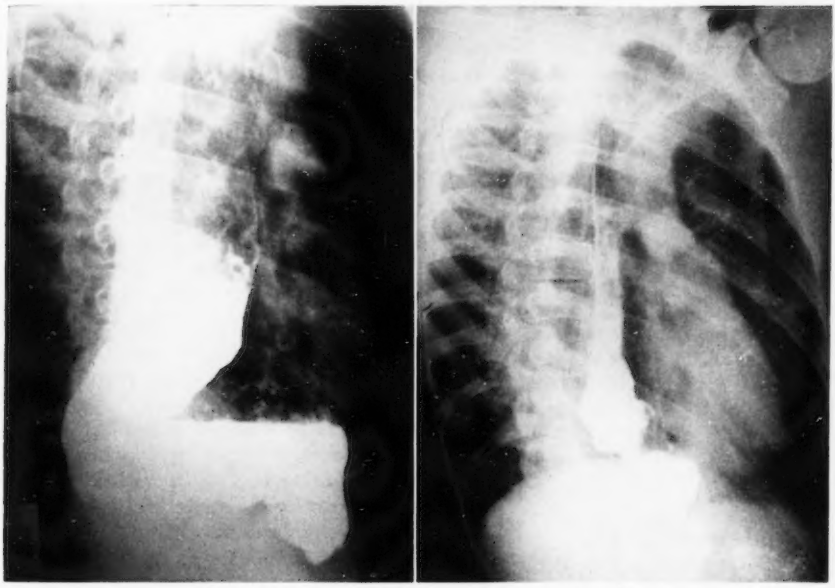
October 6 to 8: Gained 10 pounds since surgery. Eating well.

Esophagoscopy: Normal to 43 cm. No esophagitis; no reflux of gastric juice, October 7.

Esophagogram for motility on October 8: No evidence of a functional organic obstruction at the distal end of the esophagus. The barium passes freely into the fundus of the stomach. The previous evidence of esophageal dilatation has disappeared.

EXCISION OF THE ACID-SECRETING AREA TOGETHER WITH THE TORTUOUS BAG-LIKE SEGMENT IN MEGAESOPHAGUS

The discussion earlier in this presentation of the case with which esophagitis attends excision of the hypertonic segment when a good segment of the acid-secreting area of the stomach remains suggests definitely that when excision technics are employed for the relief of this type of megaesophagus, the entire acid-secreting area of the stomach should be excised. This operation has been carried out on a number of patients



a b

FIG. 13.—Case of Mr. J. B., aged 31 at the time of operation. This patient had gone through several attacks of aspiration pneumonia; there had been dysphagia for at least fifteen years prior to operation. Hydrostatic dilatation had been unsuccessful. a. The tortuous, baglike condition of the esophagus is very evident. In fact, the esophagus is boot-shaped. b. The situation four months after operation. The esophagus has practically recovered its normal size.

There have been no episodes of pneumonia since the operation was performed, a period of 30 months. The patient has regained some weight and has been completely rehabilitated.

The type of operation done here was straightening of the redundant esophagus, accompanied by excision of the entire acid-secreting area of the stomach. The antrum was then united end-to-end with the esophagus, and pylorotomy was done. This operation is illustrated in figure 10a.

in the manner indicated in figure 9a. The roentgenograms of an esophagus in which such an operation was carried out is shown in figure 13. This appears to be a very satisfactory manner of dealing with advanced cases of esophageal dystonia.

It remains to be seen, however, whether the Heller myotomy carried out with the plan herein described, employing the Bardex catheter, in which procedure complete rupture of the muscle fibers of the terminal esophagus is obtained, will be adequate for the baglike esophagus of megaesophagus. There are evidences of some tonicity in the x-ray films of the dilated esophagus of the patient, Mr. J. S., cited in the case history (fig. 12a). That circumstance may have been an important factor, too, in the quick return of the esophagus to normal size after the Heller myotomy performed with the aid of the Bardex catheter. Yet, it is to be noted that the completely atonic esophagus of Mr. J. B. had returned to normal size within four months after excision of the redundant esophagus and the entire acid-secreting area of the stomach.*

*Two additional Heller myotomies have been done employing the Bardex balloon-tipped catheter described herein since this paper was presented. In both instances, there was a prompt reduction in diameter of the dilated esophagus—an occurrence which lends credence to the thesis proposed herein that in the ordinary Heller myotomy an incomplete division of the muscle fibers is made.

SUMMARY

Lessons learned somewhat fortuitously concerning idiopathic stricture of the esophagus have served to indicate how very sensitive the esophageal mucosa is to injury by the digestive juices. Inasmuch as the mucosal lining of the esophagus is of the squamous cell variety, this great susceptibility of the esophagus to injury by acid-peptic juice in particular is immediately apparent to all students of acid-peptic ulcer. It is well known that in animals having a portion of the upper fundic pouch of the stomach lined by squamous epithelium, this area is the most vulnerable to injury by the gastric juice. Esophagitis is readily caused in the experimental animal when the digestive juices are refluxed into the esophagus. When the pylorus is obstructed in the dog so that all the gastric juice is regurgitated, evidences of severe esophagitis and perforation are very frequent. In fact it would appear that so-called spontaneous perforation of the esophagus *is* acid-peptic ulcer of the esophagus. The uniform relief obtained in instances of idiopathic stricture of the esophagus by gastric resection, in which free drainage from the stomach is also provided for, suggests definitely, too, that idiopathic stricture of the esophagus in all likelihood *is* acid-peptic ulcer of the esophagus.

There is not very much known concerning the etiology of megaesophagus. It would appear to owe its origin to absence of the parasympathetic ganglion cells in the plexus of Auerbach, located in the circular muscle of esophageal wall. There are essentially two récognizable components in the disease: (1) hypertonus of the terminal esophagus; (2) atony of the more proximal reaches of the esophagus.

The observations concerning esophagitis reported herein have been the means of teaching the limitations of many of the recently practiced operations for the relief of megaesophagus. Obviously, any operative procedure of the Heyrovsky-Gröndahl variety invites esophagitis in that reflux of gastric juice into the esophagus may occur freely.

In the baglike tortuous dilatation of megaesophagus, the writer has straightened the tortuous esophagus, excising a generous segment of the dilated esophagus together with the hypertonic area. The entire acid-secreting area of the stomach is also excised, and the esophagus is anastomosed to the antrum of the stomach. An extramucosal myotomy of the Ramstedt variety is made to ensure satisfactory emptying of the vagotomized antrum. This operation has been quite uniformly successful in the management of megaesophagus. It is not followed by esophagitis, as are lesser excisions in which a good fragment of the acid-secreting area of the stomach remains.

The only other operative procedure which would appear to have a place in the treatment of megaesophagus is the extramucosal myotomy of Heller. This operation appears not to be followed by esophagitis as regularly as occurs following the open cardioplasty of Wendel.

In a recent patient in whom a Bardex balloon-tipped catheter was employed to complete the Heller myotomy, there was early and complete return of the esophagus to normal size, a circumstance which suggests that the reduction in size of the esophagus may be dependent in some measure upon the completeness of the operation. A Heineke-Mikulicz type of pyloroplasty has been added in the Heller procedure to ensure ready and satisfactory gastric evacuation, thus decreasing the opportunity for reflux of gastric juice into the esophagus, which is the source of esophagitis following operations for the relief of esophageal dystonia.

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DISCUSSION

PAUL H. HOLINGER, M.D., Chicago, Ill.: In this most excellent presentation Dr. Wangensteen has explained the mechanism and symptom complex of one of the most serious problems encountered in esophagology today. The surgical excision of either a benign or malignant stricture obstructing the flow of food from the esophagus to the stomach would seem to be an obvious therapeutic procedure. A glance at the obstruction as it is visualized under fluoroscopy or on the x-ray film gives further weight to the impression that a simple mechanical obstruction requires only simple mechanical excision or circumvention. Yet the physiology of this area is so complex that to ignore the problem Dr. Wangensteen has presented merely leads to pathology often more severe than the original disease entity.

From the standpoint of the esophagoscopist, the problem of megaesophagus and esophagitis is a common one. We prefer to divide the cases into two groups: those without organic stricture and those in whom the original simple hypertonus of the distal esophageal segment and atonia of the more proximal esophagus is complicated by an organic stricture which is often several inches in length and sometimes as hard as cartilage. In the former type, dilatation by pneumatic bougies, we believe, is often more than palliative, as Dr. Wangensteen suggests, but is, in reality, curative. We believe that a preliminary esophagoscopy is imperative to determine the presence or absence of an associated esophagitis or peptic ulcer, and

if these are found, a week of management on an ulcer regimen may precede active pneumatic or hydrostatic dilatation. However, when the condition is found to have progressed to a megaesophagus, complicated by organic stenosis, some form of surgical assistance is indicated. The problems Dr. Wangensteen has presented account for the high percentage of patients operated upon who return in a year or two for dilatation of the postsurgical stricture. Such patients start with the severe epigastric and substernal pain and discomfort often radiating to the back, which is characteristic of esophagitis. Later a nodular, firm, tortuous stricture develops around the site of the surgical anastomosis which is exceedingly difficult to cannulate and dilate. It is a problem not only associated with the esophagitis following surgery for megaesophagus, but one which follows many of the surgical procedures for congenital esophageal stenoses, lye strictures of the esophagus, other benign strictures and carcinoma.

Therefore, we agree with Dr. Wangensteen that in cases of megaesophagus, if response to the more simple, efficient and statistically effective procedures of "bloodless Heller myotomy," i.e., pneumatic, hydrostatic or mercury bougie dilatation, is not obtained, more radical surgical measures are indicated. But we must abandon the surgical technics used for this and other stenosing esophageal lesions that rely merely on the excision of the mechanical obstruction, recognizing that such procedures destroy the physiology of the cardia. The Heller type of operation is unsuccessful if a true organic stricture is present. Vagotomy and a Ramstedt myotomy of the pylorus in association with cardioplasty are procedures also of doubtful value in such cases in our experience. Apparently a total gastrectomy, excision of the redundant terminal portion of the esophagus and esophagojejunal anastomosis by the Roux Y principle of operation are necessary to overcome the severe problem of postoperative esophagitis and ultimate perforation or stenosis that follows the simpler surgical procedure.

Dr. Wangensteen has presented an extremely important topic to us and I appreciate having an opportunity to discuss this paper.

DR. WANGENSTEEN: I appreciate very much the fine discussion of this presentation by Dr. Holinger. He speaks from an experience with megaesophagus far larger than my own.

There is only one bit of evidence that I should like to add concerning the likely verity of the suggestion that there is an important difference between the Wendel and the Heller procedures, which to many of you may not look very different. The Wendel is an open linear slit extending through the mucosa, with transverse closure; the Heller, on the contrary, is a simple extramucosal linear myotomy. Apart from the clinical information which I detailed to you, very briefly the observation which we have had the opportunity to make in the experimental laboratory is this: when the Wendel procedure is performed on dogs and esophagoscopy is carried out subsequently at intervals over a period of months, the observed incidence of esophagitis is quite high. We have yet to see esophagitis following the Heller procedure in dogs.

Perhaps I dealt a little unkindly with my colleague from Minnesota, Dr. Olsen, in my illusion to his figures but I was measuring from his base line. He reported on 555 cases but succeeded in tracing 452 of the 555 patients (80 per cent). Now, you and I know very well from our experience that a satisfied patient is an easy man to trace. I think it might be a bit unfair for me to say that all the others had unsatisfactory results. But I believe that most of you will agree with me when I suggest that a large percentage of untraced patients probably would fall into the unsatisfied group.

Among the traced cases, 60 per cent responded to one dilatation; 38 per cent of those failing to respond to one dilatation, responded satisfactorily to a second dilatation, and a very small group to the third. I would take it that even on the basis of Dr. Olsen's own evaluation of their experience, rarely is it necessary, or wise, to do more than two hydrostatic dilatations.

Sifers and Crile are far less enthusiastic over the results of treatment of megaesophagus by use of the hydrostatic dilator. They report definite clinical improvement in only 30 per cent of patients subjected to dilatation. Fritz, Clark and Adams from the University of Chicago are not enthusiastic over the results of hydrostatic dilatation of megaesophagus. And, on the basis of other reports in the literature, it is my impression that results satisfying to both the patient and his physician are obtained in approximately 50 to 60 per cent of the patients treated. Moreover, I infer that even a satisfactory result fails to achieve reduction in size of the esophagus. In other words, a satisfactory result is functionally good but physiologically incomplete. Now that we do have access to satisfactory means of surgical relief of those patients who fail to get a good result by conservative means, I do not believe that we should wait until the patient begins to fight off invalidism before operation is suggested to him.

Operations for the relief of megaesophagus in the past have not been popular, and rightfully so, for most of the operations, though followed by relief of the difficulty in swallowing, were accompanied later by hemorrhage and stricture owing

to the great susceptibility of the esophagus to injury by acid-peptic juice. During the ten-year interval of Olsen's study of the Mayo Clinic series, among a total of 601 patients classified and treated for megaesophagus during those years, only 23 (3.4 per cent) were sent for operation. One might say because hydrostatic dilatation was successful in most of the cases. Examination of the report does not warrant that conclusion. I am certain the physicians of the Mayo Clinic are not wanting in any sense of appreciation of their own surgical colleagues. On that score, there is considerable evidence to the contrary. No, I believe we can conclude that the want of confidence was in the available operative procedures employed by their surgeons. Now that we have a better assessment of what is necessary in an operation directed at the relief of megaesophagus, I am inclined to believe that the previous justifiable restraint in sending for operation patients who do not respond satisfactorily to hydrostatic dilatation, will disappear.

In subsequent conclaves perhaps someone will come forward with an explanation which will lead us, with brighter light than we have now on the subject, into a more intelligible understanding of what the true nature of megaesophagus is. We need that light so badly.

A NEW METHOD FOR OBTAINING EUSTACHIAN TUBE CLOSURE AT TYMPANOMASTOIDECTOMY

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CONTINUED discharge from an ear after radical mastoidectomy (tympanomastoidectomy) has been of concern to otologist and patient alike for many years.

Several causes have been cited as responsible for this inconvenient discharge. One author[3] lists inadequate removal of diseased bone, incomplete epithelization of the cavity with residual granulating surfaces and eustachian tube patency as the three principal causes. Another[2] states that failure to close the eustachian tube, infection of the cavity with a fungus or mold, or failure to eradicate the original infection may be responsible. A third[4] indicates that residual disease foci in the hypotympanum and inaccurate or officious surgery at the tympanic orifice of the eustachian tube are the most common causes of postoperative discharge.

Patency of the eustachian tube is common to almost all the lists. And in fact, in these days of antibiotics and improved surgical armamentarium, principally the motor-driven burr, proper illumination and magnification, all of which enable the surgeon to deal more adequately with the other causes for postoperative otorrhea, it would seem that tubal patency takes on even more importance. The problem of how to obtain permanent closure of the tube at operation is not new. Holmgren[8] stated twenty years ago that for decades otologists have been striving to close the tube to prevent tubal mucosa from covering the promontory and to prevent infection from gaining access to the middle ear region through the tube. In 1916, in an evaluation of the results of 25 tympanomastoidectomies observed for five months or longer, Harris[7] found 12, or 48 per cent, completely dry. Of those successful cases, 10 had closed eustachian tubes, while only 2 cases with open tubes could be considered successful. Attempts to accomplish tubal closure have been varied and often interesting, but none has proved uniformly successful over a long period and in the hands of different surgeons.

Gerber was the first to propose transplantation of epithelium over the orifice of the tube. Subsequently, numerous types of free and pedicle grafts were used, both from the structures in the region of the postauricular incision and from the covering of the promontory. Occlusion of the tube using several types of material was also tried. Heine suggested using the remnants of the tympanic membrane, and later used paraffin. Passow and Sercer tried plugging the tube with catgut, while Witt-

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maack and Laurowitsch used plugs of animal horn or bone. Ohngren used periosteum from over the mastoid process, covered with a skin graft. Two surgeons used galvanocautery in the tube. Almost all urged that the tubal epithelium be completely removed, and today most authors agree that the tube should be cleaned, though there is some disagreement as to whether it should be done for purposes of encouraging fibrosis to accomplish tubal closure or whether it is done simply for the removal of infected mucosa.

More recently, Goodyear[6] advocated the use of the tensor tympani muscle to plug the tube. Substances such as oxycel and gelfoam have been and still are used to occlude the tympanic end of the tube, covered then by split thickness or full thickness skin grafts. One author[9] reports good results from occluding the tube with conical plugs of polyethylene, while another[2] reports good results in 5 cases using chips of bone from the mastoid cortex.

Usually, the rationale behind the occlusion of the tube has been to stimulate fibrosis which would result in permanent closure and avoid tubal discharge, especially during upper respiratory infections. After careful removal of the tensor tympani muscle and all soft tissue within the bony tube as far as can be reached with curet and motor-driven burr, the tube at tympanomastoidectomy consists of a cylindrical defect in periosteal bone. It was conceived that this defect might be repaired permanently by plugging it with a bone graft. The most readily available source of bone would be the mastoid process, as reported by Ashley,[2] but in using that bone, two distinct disadvantages are held to be present. The first is the possibility of infection in the bone; the second is the nonporous nature of this cortical bone.

Because of its high cellular content and porosity, cancellous bone is preferred by most orthopedists and plastic surgeons for grafting. Accordingly, 5 tympanomastoidectomies were done by one of us using cancellous bone grafts to occlude the tympanic end of the eustachian tube, and a preliminary report was recently published.[1] The bone used was homogenous cancellous bone taken from a bone bank. The results were neither disheartening nor very encouraging. One of the 5 cases was lost to follow-up, while 3 of the remaining 4 cavities healed quickly and completely with closed eustachian tubes. One was a failure, having an open tube and discharge one and one-half years after operation.

Believing, however, that the idea has some merit, and since autogenous bone is known to heal more quickly than material taken from a bone bank, the work was continued with bone removed from the iliac crest of the patient at the time of the tympanomastoidectomy. It is these cases and this method that are being reported now.

Since a skin graft was used in all these cases, the technic included the preparation of the region of the anterior superior iliac spine as the donor site for both bone and skin. The draping of the patient was done in such a manner as to allow the exposure of the donor site without contamination, and the surgeon's gown and gloves were changed after the tympanomastoidectomy was finished and before the grafts were taken. The bone graft instruments are kept on a separate sterile table.

The taking of the bone graft consists simply of making the skin taut over the iliac crest immediately behind the anterior superior spine by grasping the bone through the skin and pressing a small (no. 12) blade directly down to the

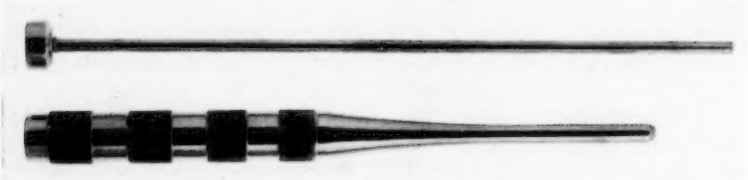

FIG. 1—The bone punch is available from the Storz Instrument Company, St. Louis, Mo. Turning the instrument as it is being driven into the bone greatly facilitates its removal.

bone. The incision thus measures less than a centimeter in length, and its direction may be parallel to the fibers of the external oblique aponeurosis or may be in the long axis of the iliac crest. Through the small incision, a small bone punch, such as is used in bone biopsies, or better still, one designed by one of us (D.A.C.) (fig. 1), is placed directly against the bone and driven in a downward, backward and inward direction for approximately a centimeter. On removal of the punch with a twisting motion and tamping out the plug with the obturator, one has a plug of bone with a bit of soft tissue and perhaps cartilage on one end which may be trimmed away to leave only cancellous bone. More than one plug may be removed from the same small incision simply by changing the direction of the punch in the plane of the wing of the ilium.

It was formerly thought that in children, due to the cartilaginous cap that remains over the crest until adulthood, it was advisable to procure the bone through the lateral surface of the ilium. In all of the cases being reported, however, the same approach was made with no regard to age. The youngest in this series was 12 years of age, and several others at operation had a thin cap of cartilage which was pierced to obtain the bone. No untoward results were noted, and in fact none were expected since there are no articular surfaces adjacent to or near the donor site.

One stitch usually suffices to close the incision through which the bone has been taken, and the skin graft may be taken from the same immediate area. Slightly more difficulty has been encountered in taking the skin graft from just below the iliac spine than from the femoral surface of the thigh; however, enlargement of the prepared area will permit using a more inferior site if desired.

The slicing of the cylindrical bit of bone into thin discs, or otherwise rendering it into mush or small pieces, is then carried out so that the cleansed tube may be firmly packed (fig. 2). The packing of the eustachian tube should be carried out only to the level of the anterior tympanic wall so that a smooth surface may be presented for the application of the skin graft. Spaces between the bone discs are avoided by the use of an instrument for packing, such as the blunt end of a metal applicator or the back of a mastoid curet cup.

Admittedly this work has been too recently done and the number of cases is as yet too small for broad conclusions to be reached. Much work is still left to be done. For instance, we do not know the exact fate of the bone placed in the tube, though we hope that it becomes an integral part of the temporal bone as bone grafts are known to do elsewhere, and that it serves to close the tube permanently. In most of the cases to be re-

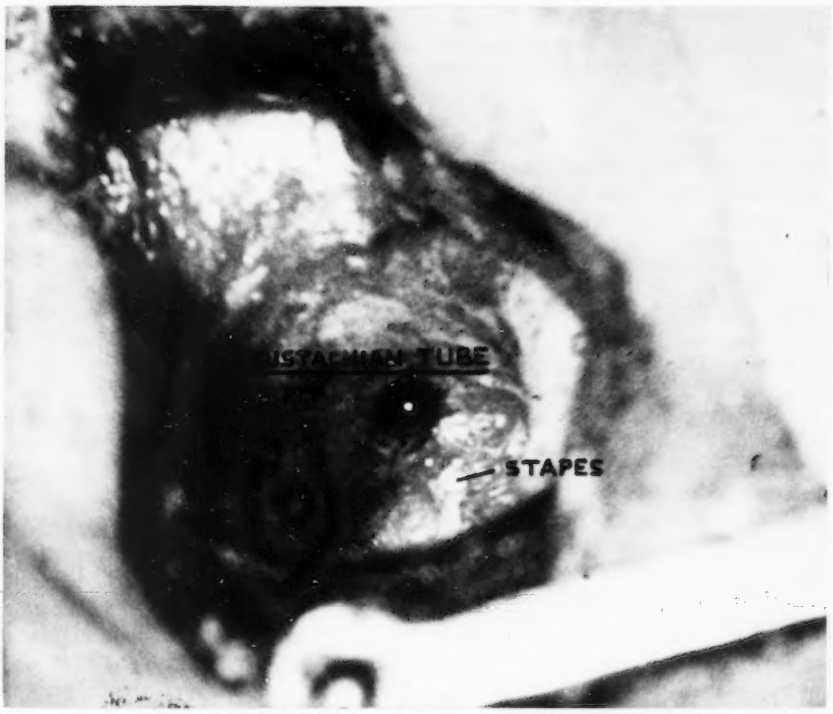

FIG. 2.—This photograph demonstrates that after adequate removal of the annulus tympanicus, outer attic wall and part of the tympanic plate, the eustachian tube is readily accessible for the placement of the bone graft.

The capitulum of the stapes and the stapedius tendon are visible in the posterior part of the tympanic cavity.

ported, we do know that the tube is closed and the skin graft well taken in the middle ear, which is after all our objective.

RESULTS

In the evaluation of our cases, the definition of a "dry ear" is one in which the cavity is completely lined with squamous epithelium and the eustachian tube is closed. No mucosa-covered areas or granulations are present, including the inner aspect of the incisional area which, in our experience, is the last to heal. The ear does not become wet with upper respiratory infections. The lone exception is the occasional patient with a neglected cavity who appears, usually during the summer months, with a moist mass of desquamated epithelium and wax present. Following removal of the mass and the drying effect of a 95 per cent alcohol application, the cavity again meets the above specifications.

During the past eighteen months, a total of 23 ears in 22 patients have been operated upon by us using this method. Eight of these were primary operations, while fifteen were revisions. Three of these revisions had had three previous operations each. Twenty-two of the cases were seen by us for final

evaluation during a six weeks' period immediately preceding the writing of this report.

Of the 8 primary cases, 6 are totally dry, and 2 have not had sufficient time for total healing. Both of these have closed eustachian tubes and completely healed middle ears, and we feel that we may reasonably expect them to progress to complete dryness, especially since the only remaining granulations are just within the endaural incisional area.

Of the fifteen revisions (problem cases), fourteen have been available for follow-up. All of these have closed eustachian tubes to visualization and taste testing, using warm 95 per cent alcohol in the cavity with the head turned to the opposite side and including the Valsalva maneuver, during which bubbles may be seen if the tube is patent. It is in this group that we report three failures.

Certainly other factors than tubal patency may be responsible for failure to obtain totally dry ears, both at primary and revision operations. Some of these factors are persistent mucosa in unexenterated cells or in the difficult to cleanse sinus tympani, the occasional presence of fungus or mold, and allergy. We have considered allergy as possibly responsible for some of these cases in the group now being presented and in others in our series, and we have undertaken allergic management of them. When tubal patency is present, as well as mucosa or granulations in the cavity, it seems logical that at revision one may hope for good results by closing the tube and replacing persistent mucosa, which may serve as a sensitive "shock organ," with squamous epithelium, which is thought to be less sensitive.

Eight of the revisions are totally dry. Four have had insufficient time for complete healing, but at the time of writing had closed tubes and completely epithelized middle ears. One of the 14 has a small persistent granulation on the medial wall of the middle ear in its lower part, while a second has a small moist area in the sinus tympani. The third failure was later revised and the failure properly attributed to faulty operative technic since the upper one third of the tube was found to be open and filled with granulations which communicated with granulations present in the hypotympanic portion of the middle ear. The granulations in the tube and the tract of granulation tissue communicating with the granulations in the hypotympanic area existed under dry intact squamous epithelium covering most of the medial wall of the middle ear and the eustachian tube. We consider this finding significant. Therefore, there are three failures, and we suspect that in the two unrevised ones we may later find partial or complete patency of the eustachian tube since we have found it in the case just mentioned and in two previous revisions on cases done prior to the bone graft method. In all 3 of these cases, the tube was covered with intact dry squamous epithelium and appeared to be closed to visualization and to our other tests. Incidentally, the one failure which has been revised has been reclassified as a success.

The healing time has varied from less than 35 days to less than 97 days, and these numbers indicate the actual number of days after the operation to visual verification of a totally dry ear by one of us. Several of the patients have reported that their ears had been dry for a number of days or weeks before this. It has been noted in these bone graft cases that the middle ears have healed with rapidity, being almost always completely epithelized at the end of three weeks when the cavities are first completely cleansed. The undersurface of

the incisional area proved to be responsible for more than half of the total number of days required for the cavities to attain a state of total dryness.

We believe the results which we have obtained on the primary cases to be very encouraging. Although the number is small, we believe that the method is worthy of further study and of recommendation for trial. We also feel that the good results obtained in the primary cases may be in part attributable to the fact that the usual landmarks were not obscured by previous surgery. It has been our feeling, substantiated by our results, that the best chance for complete tubal closure and a dry ear is at the first operation.

The revisions represented those problem cases in a series of over one hundred mastoid operations in which the patient was willing to return to the hospital for another attempt to secure a dry ear. Persistent pain and vertigo were also considered indications for revision. All the revision cases which were done initially by either of us had had curettage of the eustachian tube and gelfoam packing, followed by a split thickness skin graft. Four of the revisions had positive fistula tests before their revision, and their discharge appeared to have some effect upon producing their vertiginous complaints. All four of these now have dry cavities.

COMMENT

In all of our mastoid operations, the split thickness skin graft is used. We feel that it contributes much to the rapid epithelization of the cavity, avoiding the development of synechias and obviating the necessity for postoperative packing. Realizing that the thickness of a graft depends upon the nutrition offered by the recipient site as well as the function to be demanded of the graft, then the thinner the graft, the more successful it might be expected to be in a mastoid cavity, since little nutrition could be expected from bone and since no function except coverage is required. The outer cells in the horny layer of the skin are accustomed to survival by diffusion of nutriment from a distance, so a very thin graft is desirable.[5]

Accordingly, an application of 5 per cent gentian violet in alcohol is first applied to the donor area. This is followed by a coating of thick collodion. A free-hand graft is taken with a Ferris-Smith skin graft knife; this is as thin as possible, even to the extent that small perforations appear. No hair follicles are included, of course, due to the superficial nature of the graft. The collodion facilitates the handling of the graft and the gentian violet positively identifies the outer surface. The graft is cut into smaller pieces and the entire cavity carefully lined, with as little overlapping as possible. On the twelfth postoperative day, the donor site, as a rule, is dry and requires no further dressing.

The revision cases have provided a difficult test of the method, but we believe at the present time that the results are encouraging. However, the small number of cases renders any conclusion subject to certain reservations, including observation over a longer period.

Our experience in revising one of our failures, and in two other revisions without bone grafts, has led us to suspect the presence of an open tube when granulations or mucosa are seen in any part of the middle ear. A tract of granulations, or an actual fistulous tract, may travel for some distance in the middle ear under cover of normal-appearing squamous epithelium. And, based upon these three case observations, we think that even after the middle ear is completely healed, if the tube is not firmly occluded, breakdown of the squamous

epithelium may occur elsewhere in the middle ear than directly over the tube. Therefore, we are more and more convinced that osseous closure of the tube is desirable.

This method of occluding the bony eustachian tube with cancellous bone is presented as an adjunct to properly done tympanomastoidectomy. It is no substitute for meticulous cleansing of the middle ear under magnification. In fact, its successful employment requires careful removal of the tensor tympani muscle followed by curettage or burr-cleansing of the bony tube, which we believe facilitates the adoption of the bone graft.

The taking of the bone graft itself is a matter which will add no more than ten minutes to the operating time, including preparation and draping of the donor site and the suturing of the incision for obtaining the bone. It has proven, in our experience, to be particularly valuable for the problem cases, patients who have had one or more operations and who still are plagued with presistent discharge due to tubal patency, with or without pain or vertigo. In the final estimate, regardless of the care with which one explains to the patient that the operation has rendered his ear "safe," if not absolutely dry, he is still apt to judge the success or failure of the procedure upon the presence or absence of discharge.

SUMMARY

The results in 22 cases of tympanomastoidectomy using autogenous cancellous bone to obtain permanent eustachian tube closure are presented. The bone is obtained from the iliac crest, using a small bone punch, and no untoward effects from this procedure have been noted. The method in our hands has given good results in primary tympanomastoidectomies and offers much promise in problem revision cases, though the number of cases and the short postoperative time do not allow broad conclusions to be reached.

Addendum No. 1

Since the preparation of this manuscript, the authors' attention was called to an article in the September 1952 issue of the *Journal of Laryngology and Otology* by Dr. Anthony Radcliffe of London in which he describes almost identically the same procedure, using bone procured from the ilium in a similar manner and for the same purpose, and gives his results obtained over the period since 1947. Acknowledgment of his priority in the method is heartily given. This work was begun independently by the senior author in 1950.

Addendum No. 2

One of the cases which has been reported as having persistent granulation tissue in the hypotympanum has been revised since the completion of this report. A nidus of osteomyelitic bone in the hypotympanum was found and removed. The eustachian tube region was explored. The bone graft was found to be quite firmly in place, and the bone to all appearances was living and well "taken."

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DISCUSSION

PHILIP E. MELTZER, M.D., Boston, Mass.: The problem to which this paper suggests a solution has been with us for a long time—in fact, since radical mastoidectomy was first performed. Each succeeding generation of otologists has struggled with it and has come up with its own solution. But the problem is still with us, although many of the factors that have in the past given rise to it have, over the years, been eliminated for the most part by the advances in surgical technics and in postoperative management. Helpful as many of these new technics have proved to be for specific problems, the fundamental need is not for new technics but for more able surgeons. As surgeons are given more and more opportunities to improve their technical skill and increase their knowledge of temporal bone anatomy, the postoperative discharging ear will become less and less of a problem.

And now, to consider first things first, let us begin with what we can presume to be the causes of middle ear discharge following radical mastoidectomy. The authors have enumerated the major causative factors. Most of them revolve around the following:

1. Incomplete epithelization or a breakdown of epithelial lining of unknown cause, similar to breakdown of epithelium anywhere in the body, possibly due to allergy

2. Bacteria or fungus (or mold)

3. Inadequate surgery of foci in any part of the temporal bone, or inadequate postoperative care

4. Reinfection of the tympanum from ascending infection through the eustachian tube

The writers define a dry ear as one in which the cavity is completely lined with squamous epithelium and the eustachian tube is closed. I take issue with this definition. Many cavities are, in my experience, completely epithelized and dry, yet the tube is still open. In these cases the eustachian tube was never a factor in producing the chronic condition in the ear in the first place. Experience leads me to believe that intermittent or continued discharge in the middle ear, as differentiated from discharge elsewhere in the mastoid cavity, is usually the result of incomplete surgery to the middle ear and tubal orifice. It may, however, also be due to infection of upper air passages. This is the type of case in which continued discharge after complete middle ear surgery may be due to ascending tubal infection. A good history, eliciting the fact that the discharge begins and increases with the infection and diminishes rapidly with the clearing of the infection, will localize the source of the trouble.

I am sure the authors are not implying that breakdown of the epithelial surfaces of the tegmen or mastoid cavity is the result of patency of the eustachian tube. We have all seen cavities that have been dry for years in which, for some reason or other, areas—sometimes small, sometimes large—become denuded of epithelium and begin discharging. The discharge may persist for some time but generally clears up promptly with local treatment.

It is quite evident to me that the authors have developed a skill in temporal bone surgery which enables them to explore thoroughly the hidden recesses of the tympanic cavity. I have a feeling that the cavities in most of their cases would be dry even if the bone graft were not applied. I find that when the surgery to these areas is thorough enough, tubal discharge is a rare—and I mean rare—complication. This is true only if you have excluded acute infection or exacerbations of chronic infections originating from the nose or nasopharynx. With a negative history of such ascending infections you can be assured that, in spite of the competence of the surgeon, the operative procedure was inadequate. No surgeon is infallible in this respect.

Let us consider now the specific technic recommended in this paper. My experience with attempted closure of the eustachian tube in the past has included everything but bone. This method of bone grafting is entirely new to me. The authors have proved that it can be used effectively to close the eustachian tube, and I concede that in certain cases this procedure may have merit.

There are many reasons why the radical mastoidectomy is not completely successful, as far as discharge is concerned, but this is a subject in itself and not pertinent to the paper under discussion.

It would be interesting to know what the cause for the continuing discharge was found to be in the 15 revisions performed by the authors. Was the cause failure to eradicate the disease at the first or subsequent operation, in other words inadequate surgery?

As to the final results, the number of cases are rather limited and are too few for me to comment upon. All I can say is that the authors have accomplished what most experienced surgeons are able to do without the bone graft. The bone graft apparently has no influence on the healing time of the cavity, as it takes about as long as other technics.

The question finally resolves itself to the real value of obliterating the tympanic end of the eustachian tube. It has been a rule of mine in surgery of the temporal bone to do the surgery as completely as the given case justifies. To do more than what is necessary has always seemed to me to be unwise. On the other hand, I am not so completely a slave to habit but what I can adopt and apply new technics when they are called for. I will, however, concede that in certain situations where the history indicates that the discharge in the middle ear is definitely the result of upper respiratory infection ascending the tube, this procedure—as the writers suggest—might be tried.

What pleases me most is that here are young men who are thinking in terms of further improvement of our results. They followed through with an idea which at first was not too encouraging. This is the way progress is made. If, in my discussion, I appear to be skeptical, this skepticism is intended to caution not the authors but those of you who think that a bone graft in the tube obviates the necessity of detailed and complete surgery to the middle ear space. I will await with interest their later reports.

PERCY E. IRELAND, M.D., Toronto, Ont. Canada: My old teacher, Dr. Meltzer, has said most of the things that I would wish to say about this method of tube closure, because I learned a good many of my surgical principles in ear work from him.

I should like to say that I was given a copy of this paper ahead of time. I had an opportunity to read it and enjoyed it very much.

I should like to emphasize one thing that Dr. Meltzer said; that is, we cannot stay in a rut. We have to have new ideas and we have the younger people coming along with the new ideas. We cannot always be correct ourselves in our old and staid methods of treatment.

I should like to hear further about this method. I do not think it is a good thing for everybody to try it in all cases.

I suspect that I was asked to discuss this paper because of some of the peculiar mastoid cavities that were seen by members of the American Board and by candidates when they were in Toronto in May. They are certainly much more peculiar than the mastoid cavities that Dr. Meltzer is speaking about. They represent the ideas of a young man, and I think they are not without merit. I am not trying to advise competition in new methods, but if Dr. Douglas Bryce is here, I should like him to take two minutes, with your permission, to tell why and how we had those peculiar, radical mastoid cavities that you saw in Toronto.

I may say that Dr. Bryce is one of our junior staff men. He is trained in general surgery and in plastic surgery as well as in otolaryngology, which perhaps brings a little fresh blood into the specialty. He is also a candidate for fellowship in this Academy.

DOUGLAS P. BRYCE, M.D., Toronto, Ont. Canada (by invitation): The work to which Dr. Ireland has referred and which was demonstrated last spring was started about three years ago. The purpose was to be able to guarantee to a patient a dry ear and, secondly, to be able to eliminate postoperative care. We had in mind particularly patients who might not be able to return to a center to have the necessary postoperative care of their ears. Obviously, the selection of his very important cases and the operation is undertaken only in those ears in which we feel the improvement of hearing is not a factor.

The use of temporal muscle was suggested to us by the use of muscle elsewhere in surgery to overcome chronic infection, such as the use of a muscle graft in an empyema cavity or the saucerization of a tibia for osteomyelitis. We felt that the greatest value of the temporal muscle flap would be its good blood supply.

The type of case on which this method has been used is the ear which has been running for many years, where there is destruction of the landmarks in the middle ear, where there is no useful hearing, and, in particular, in those cases in which the inner ear has been destroyed by labyrinthotomy or by disease.

Originally we operated with the postauricular incision. Eventually we found that we could get at the temporal muscle better with an endaural incision. It is necessary first of all to repeat that the most important feature is a good mastoi-

dectomy. We found that the removal of the complete mastoid tip was essential. All the mucous membrane in the middle ear must also be removed meticulously. Tuck the mucous membrane that you cannot remove into the tube and remove all the skin of the external canal.

The temporal muscle is then elevated and a section is elevated and inserted into the cavity. Sometimes it is difficult to put the temporal muscle into the cavity and the posterior roof of the zygoma has to be chiseled away to allow it to come properly down into the middle ear. The muscle is held in place in the middle ear by two or three subcutaneous sutures. Originally we then tucked a split thickness skin graft in just to the meatus, in other words, obliterated the ear completely. Recently I think a flap from the floor of the anterior wall of the canal can also be utilized to close the external meatus.

There are two criticisms, of course. First, the hearing. Ability to hear a conversational voice at about two feet is about all that can be expected following operation. Secondly, the question of whether the eustachian tube will be closed. That we can only tell by results. I think a muscle flap carrying its own blood supply is probably a very effective way of closing such a tube.

We have had 10 cases, the oldest one three years ago. We have had 9 cases which healed by primary intention and were discharged from the hospital four or five days postoperatively. They required no attention whatsoever, and have had no trouble even with recurrent upper respiratory infections. We have had one failure in which a sinus developed from the middle ear.

New Instruments

ABSOLUTE THREE DIMENSIONAL COLORED RETINAL PHOTOGRAPHS

HERMAN J. NORTON, JR., M.D.
ROCHESTER, N. Y.

PHOTOGRAPHIC recordings of pathologic processes are accurate and complete only when the picture is colored and includes the third dimension. The highest degree of accuracy is obtained when the two dissimilar frames are exposed simultaneously because then the method is an absolute one rather than merely a relative one. The desirability of such an absolute stereophotograph is particularly important in the case of internal ocular photography. I wish to present a photographic unit for obtaining simultaneous-absolute three dimensional 35 mm. colored transparencies of the human retina (fig. 1).

1. *Adaption:* Two binocular instruments, the Stereo-Realist Camera and the Binocular Ophthalmoscope, are adapted to each other by two ocular tubes. Neither the ophthalmoscope nor the camera is permanently altered.

2. *Illumination:* The standard 6-volt 15 C. P. lamp of the ophthalmoscope can be used by overloading the lamp at the time of exposure. To do this readily I use an electric train transformer, which permits a wide range of voltage points, low for focusing, and high for overloading at the time of exposure.

Stronger lamps are under investigation at the moment. Slight modifications in the lamp housing will be required before a stronger lamp can be used. With the standard lamp, however, no alteration in the lamp housing is required.

3. *Magnification — Field Coverage:* The magnification is identical and the diameter of the field covered compares very favorably with that of the elaborate retinal cameras. The field coverage just about fills the complete 22 by 23 mm. stereoscopic frame of the new 335 Kodachrome film introduced by Eastman Kodak Company a few weeks ago. The Eastman Kodak Company is now offering a processing and mounting service for the new 335 stereokodachrome.

4. *Viewing Projecting Films:* The colored transparencies can be viewed with the Stereo-Realist viewer and, in addition, the same slide may be very conveniently projected with the Stereo-Vivid Projector of the Three Dimension Company.

SUMMARY

A three dimensional photographic unit for obtaining simultaneous-absolute 35 mm. colored transparencies of the retina which can be projected upon a screen has been introduced.

It is felt that the unit is very practical because of the following features: (1) It is composed of instruments readily available on the current market. (2) The adaption necessitates only temporary alteration of camera and oph-

From the Department of Surgery, Division of Ophthalmology, University of Rochester School of Medicine and Dentistry.

Presented as a *New Instrument* at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

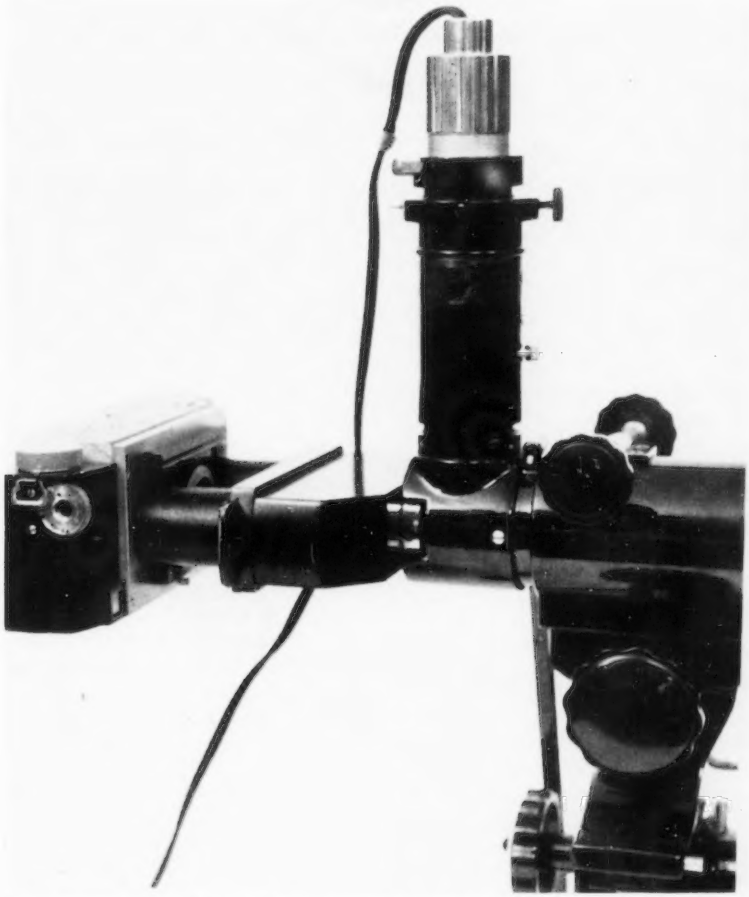

FIG. 1—Stereo-Realist adaption for simultaneous-absolute three dimensional photography of the human retina.

thalmoscope permitting each to be used for other purposes if so desired. (3) The pictures are made on the new standard stereokodachrome film recently introduced for three dimensional work by Eastman Kodak Company. (4) The adaption does not require elaborate accessories, which is not usually the case in highly specialized photography. (5) It is the only photographic device at the present time which permits taking absolute stereokodachromes of the retina. (6) The photographs obtained compare very favorably as to degree of magnification and extent of field coverage with the pictures obtained by other photographic units and in addition, the films are three dimensional.

INTRACAVITARY OPTIC RADON APPLICATOR

JOHN W. HENDERSON, M.D.
ROCHESTER, MINN.

OCCASIONALLY, in unilateral retinoblastoma in which the neoplasm is discovered early, and in bilateral retinoblastoma in which the diagnosis has been established by removal of one eye and the neoplasm is not far advanced in the second eye, irradiation therapy has been utilized to destroy the malignant tumor. This irradiation can be administered in the form of fractionated roentgen rays, such as has been widely used by Reese, Merriam and Martin,[5] by the application of radium to the ocular adnexa of the affected eye, or by the application of radon seeds directly to the surface of the eyeball. In the past several years my colleagues* and I have been interested in the latter method of therapy because it allows a more direct concentration of the gamma rays over the site of the neoplasm.

The irradiation of retinoblastomas with radon seeds is not new. In 1931 a technic was described[4] by which radon seeds were inserted directly into the neoplasm or sutured to the sclera overlying the retinoblastoma. In 1934 Barkan[1] described a technic of embedding the radon seeds in dental compound and then applying them to the eye. In 1935 Moore,[3] who had been one of the first to utilize radon seeds, told of further modifications in applying this form of therapy to the eye. Waldman,[7] in 1940, described an applicator made of rolled sheet silver or platinum into which the radon seeds could be inserted. This band-shaped applicator could then be attached to the eyeball with sutures. In 1946 Stallard[6] summarized in his textbook his experiences with various surface applicators, and detailed his present methods of therapy with radon seeds.

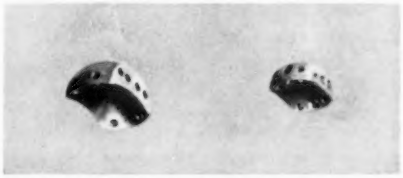

FIG. 1—Intracavitary optic radon applicators: left, large applicator; right, small applicator.

The instrument that has been in use at the Mayo Clinic for the direct application of radon seeds to the surface of the eyeball is named "intracavitary optic radon applicator"[2] (fig. 1). The instrument is essentially a rectangular, concave-convex, brass plaque with perforations provided for the insertion of radon seeds. This applicator is made in two sizes, the specifications of which are given in table I.

The perforations provided for the insertion of the radon seeds are 1 mm. in diameter. After the radon tubes are in place, the perforations can be sealed by means of a drop of melted paraffin. At each end of the applicator additional perforations are provided for the passage of sutures. The curvature of the convex surface has been calculated to

From the Section of Ophthalmology, Mayo Clinic.

*The development and use of this instrument have at all times been a cooperative undertaking in conjunction with Dr. R. E. Fricke and Dr. Martin Van Herik, of the Section of Therapeutic Radiology, and Dr. M. M. D. Williams, of the Section of Biophysics, Mayo Clinic. Blueprints of this instrument will be furnished upon request.

Presented as a *New Instrument and New Technic* at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

TABLE I

SPECIFICATIONS: INTRACAVITARY OPTIC RADON APPLICATOR

	Dimensions, mm.		No. of radon tubes	Filtration	Distance from radon to applicator surface, mm.
	Over-all	Active area			
LARGE APPLICATOR	7 by 11	5.5 by 5	4	0.3 mm. gold 0.5 mm. brass	1
SMALL APPLICATOR	6 by 8	3.5 by 4	3	0.3 mm. gold 0.5 mm. brass	1

permit close approximation to the surface of the eyeball.

The applicator is applied to the eye while the patient is under the influence of general anesthesia. One or more of the extraocular muscles may be temporarily detached from its insertion into the sclera if it will facilitate manipulation and suturing of the applicator. A treatment period of two to six hours is preferred. Anesthesia of the patient is maintained by a combination of basal sedation and small doses of pentothal sodium administered intravenously.

The technical factors concerned in the administration of a therapeutic amount of gamma irradiation have been detailed in a previous paper.[2]

A minimal tumor dose of 2,000 gamma roentgens is administered if subsequent external radium therapy is anticipated, and a minimal tumor dose of 3,500 gamma roentgens is given if additional external therapy is not to be given.

The advantages of an applicator of this type are (1) it is possible to "pinpoint" the area of therapy rather than to irradiate the entire posterior segment of the eye; (2) the treatment time is reduced to a matter of a few hours; (3) the dangers of scatter emanations are reduced to a minimum; (4) the dangers of changes in the skin subsequent to the external applications of radium are eliminated, and (5) the chances of the development of cataract are decreased.

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PERIMETRY WITH ULTRAVIOLET (BLACK) LIGHT AND LUMINESCENT TEST OBJECTS:

A Preliminary Report

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ONE of the most difficult problems in perimetry has been the elimination of distraction in the examination. Test object carriers, the examiner's hand and clothing, light reflected from marking pins, chalk and perimeter arcs plus extraneous room light, all distract the patient's attention from both the fixation target and the test object in the peripheral field, thus adding immeasurably to the difficulty of performing the examination and detracting from its accuracy.

The examination of the visual field by perimetry is a subjective test. It is the patient's interpretation of what he sees which determines the accuracy and validity of the test and it is the visualization of the test object in relation to the fixation target which determines the extent of the visual field as interpreted by each individual patient. Any method which simplifies the test for the patient by eliminating distracting stimuli from within the field of vision so that only the fixation target and the peripherally located test object are visible will materially enhance both the accuracy and ease of performance of the examination.

The numerous devices which have attempted this objective are mute testimony to its importance. A few of these are (1) the use of nonreflecting surfaces of black or grey felt on tangent screen or perimeter arc; (2) black gowns and black sleeves and gloves to be worn by the examiner, and (3) projection of the target as a spot of light on the tangent screen or perimeter arc.[2]

Of all these methods the last is the only one which successfully eliminates the distracting elements usually encountered in the examination, and it becomes impractical due to the elaborateness and considerable cost of the projection equipment required plus the fact that the accuracy of the patient's central fixation is difficult for the examiner to observe.

All of these problems are practically and economically solved through the use of "black light" and luminescent test objects in the perimeter and tangent screen examination of the visual field. With this method distracting elements are almost completely eliminated from the field, leaving only the fixation target and test object visible to the patient. Total darkness is not required and the patient's fixation and reactions may be constantly observed by the examiner. The equipment required is simple and inexpensive and, in the case of the tangent screen, utilizes existing and commonly used materials.

The Harrington "Black Light Perimeter" (figs. 1, 2 and 3) and "Lumilamp Tangent Screen Light" (fig. 4) used with the Harrington "Lumitest

From the Division of Ophthalmology, University of California Medical School, San Francisco, California.
Presented as a *New Instrument* at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

Objects" constitute the first practical application of ultraviolet light and luminescent test objects in the examination of the visual field.

Both the "Black Light Perimeter" and the "Lumilamp Tangent Screen Light" emit ultraviolet light below the visible spectrum plus a slight amount of visible blue light.

The perimeter's source of ultraviolet light is a cold cathode tube of 18 mm. diameter, rigidly suspended slightly above and 1.5 inches in front of the perimeter arc face. The tube parallels the perimeter arc for its full length so that the intensity of the light source remains constant through the entire 180 degrees.

The tube contains B-10, a commercial conducting gas whose components are argon, neon, krypton and helium plus a minute quantity of triple distilled mercury. When voltage is applied to the tube electrodes, the B-10 gas is ionized and the resultant heat vaporizes the mercury. The ultraviolet light thus generated by the ionized B-10 and vaporized mercury is amplified by ultraviolet-emitting phosphors with which the inside wall of the tube is coated. As only a portion of the ultraviolet light produced by the tube is within the band useful for purposes of visual field examination, the glass of the tube is tinted to a shade which filters out all of the radiation except that between 3200 A (Angstrom units) and 3800 A. The peak of radiation is at 3654 A. The visible spectrum begins at 3876 A.

The light tubes are standardized so that each tube emits the same lumens per foot directed against the perimeter arc surface with an even intensity over the entire 180 degrees. The estimated life of the tube is 15,000 hours.

The "Lumilamp Tangent Screen Light" employs a similar light source of much smaller size mounted in a convenient hand-held housing. The examiner directs its beam against the screen so as

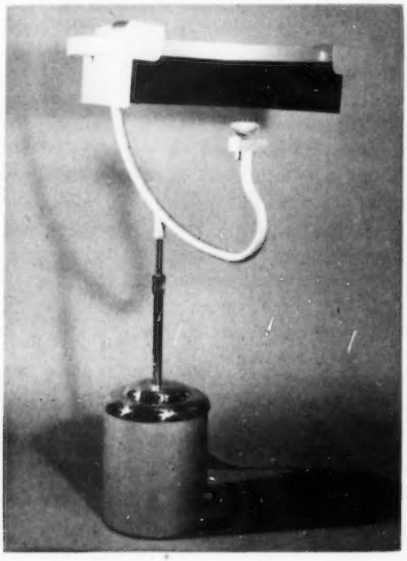


FIG. 1—Perimeter, front view, in horizontal position.

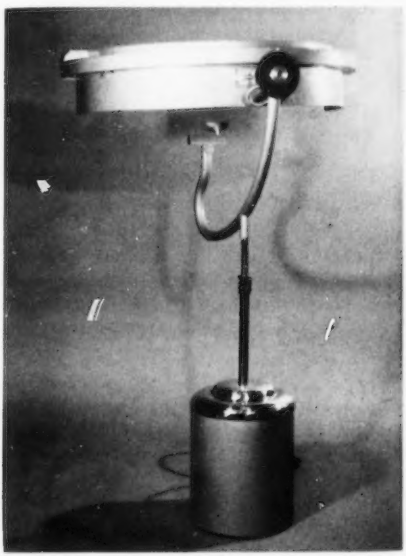


FIG. 2—Perimeter, rear view.

FIG. 3—Perimeter, front view, vertical position, for use or storage in minimal space.

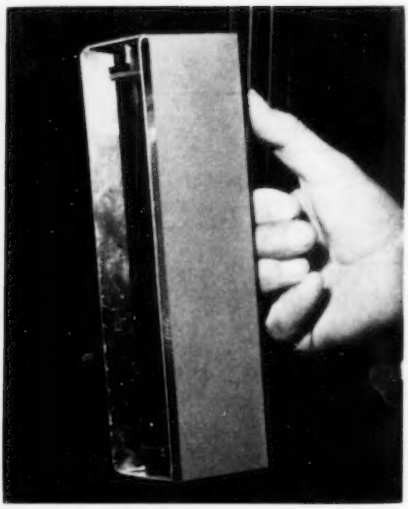


FIG. 4—"Lumilamp." Ultraviolet light source for tangent screen perimetry.

to illuminate the fixation point and the test object at the same time. The portability of the light source makes it possible to control the illumination at all times and to vary its intensity at will for greater sensitivity in the test.

The "Lumitest Objects" are luminescent sulfides which, when exposed to the ultraviolet radiation of the perimeter or tangent screen "Lumilamp," absorb the "black light" quantitatively and transform this invisible radiation into white, red, green or blue colors, depending on the sulfides used. There is no heat attendant with the phenomenon and the amount of visible light released by the targets when excited by the "black light" is relatively high.

The amount and type of luminescent sulfides used in the test objects is carefully controlled so that each test object will release the identical amount of light of exactly the same wavelength as its corresponding master test object maintained as a control by the manufacturer.

To heighten the contrast between the test object and the background and to eliminate completely the possibility of any ultraviolet radiation reaching the patient's or the examiner's eyes, the perimeter arc face and all surfaces of its base and supports are painted with enamels containing an ultraviolet absorbing compound which removes the possibility of any ultraviolet light reflection. The felt of the tangent screen may be sprayed with a colorless solution of the same ultraviolet absorber.

Since all of the illumination from the light source is below the visible spectrum with the exception of that light transformed by the test objects into visible rays of white, red, green or blue, the test objects themselves appear to the patient as the "source" of the light rather than as the reflectors of remote illumination. All sources of distraction fade into

blackness and the test objects become dots of pure light viewed against a perfect void.

Without the confusing reflection of light from the surface of the perimeter arc, tangent screen, test object carrier, test object pins, etc., a 0.5 mm. white "Lumitest" object may be quickly and accurately detected by the patient the moment it enters the limits of its isopter, and it is quite possible to sharply delimit a minute scotoma in the central field with a 0.25 mm. test object at a distance of 2 meters, a feat impossible to duplicate with reflected light scotometry except with the most intelligent and observant patient.

The Harrington Perimeter* is manufactured with either ultraviolet or visible light illumination. If standard illumination is desired for use with standard reflecting test objects, the cold cathode tube is coated so as to emit a white light of 4500 Angstrom units with an intensity of 7 foot-candles over the entire perimeter face. The standard spherical test objects of Berens et al.[1] may be used with this instrument.

The instrument satisfies the primary requirements of a practical arc perimeter with a number of improvements over existing models.

1. It has an arc whose radius of curvature is 330 mm. and of sufficient width to simulate a portion of a hollow sphere and thus allow for a degree of scotometry within the confines of this width without rotation of the arc.[3]

2. Illumination is uniform and constant on all portions of the arc.

3. The arc and illuminating system is mounted on an adjustable microphone stand, thus eliminating the need for a table and allowing for easy adjustment to varying heights for greater patient comfort.

4. The arc surface of the perimeter is covered with felt or ultraviolet absorbing dull black paint, dependent on the model used, to minimize reflection.

5. The illuminating tube is parallel to the perimeter arc so that when the arc is in the vertical position the entire instrument requires a minimum of storage space when not in use.

6. Except for the weighted base, which increases stability, the perimeter is constructed entirely of aluminum and may be wheeled on its casters from one room to another with little effort. It does not require a separate "perimeter room" for its use.

7. Standard Berens' test objects[1] may be used with the white light perimeter. A complete set of "Lumitest Objects" plus a dull black ultraviolet absorbing wand to carry the objects is included with the "black light" model.

8. The instrument is less expensive than most comparable models currently on the market.

* The Harrington Perimeter, "Black Light Perimeter," "Lumilamp Tangent Screen Light" and "Lumitest Objects" are manufactured by Jenkel-Davidson Optical Company, 366 Post Street, San Francisco, California.

REFERENCES

1. Berens, C., Kern, D., and Evans, J.: White and colored spherical test for use in campimetry and perimetry, Tr. Sect. Ophth., A.M.A., 1931, p. 407.
2. Evans, John N.: The perimeter spot-light object, Arch. Ophth., n.s. 7:614-619 (April) 1932.
3. Traquair, H. M.: An Introduction to Clinical Perimetry, ed. 3, St. Louis, C. V. Mosby Co., 1938.

"STERETAINER": A NEW DEVICE FOR DISPENSING STERILE OPHTHALMIC SOLUTIONS

RICHARD C. TROUTMAN, M.D.
NEW YORK, N. Y.

THE MANHATTAN Eye, Ear and Throat Hospital has used individual disposable ophthalmic solutions for more than five years. Recently, in conjunction with The Selney Company, we developed a new type of dispenser (fig. 1). This device has some distinct advantages over the type previously used and reported elsewhere, and so we felt it might interest others in the profession. Our prime objective in developing the Steretainer was to prevent and control infection in operative cases. Due to the large numbers of patients on our pavilions, it is essential to prevent or immediately isolate infected or potentially infected eyes. Of especial importance also is the maintenance of absolute sterility of fluorescein solutions because of the large number of cases of corneal surgery. These problems have been solved with the Steretainer, which may be cold sterilized for operating room use or used as an individual disposable unit on the pavilions. All solutions in use at our hospital for preoperative, operative and postoperative medication are made available in this unit.

The device is so made that a perfect seal is obtained. The contents are clearly marked in a visible and insoluble ceramic ink which provides a distinctive color as well as the name and percentage for each solution. A special rubber is used which is chemically inert and which, according to manufacturers, actually aids in preservation of the solution. A rubber valve is built into the bulb to prevent the solution from entering this bulb from the tube and also to prevent release of the contents when the rubber tip is removed, which might occur because of the sudden change in atmospheric pressure. The solutions used are especially buffered and will, according to other reports, retain their effectiveness longer than the usually prepared ophthalmic solutions.

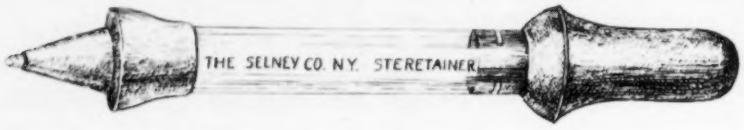


FIGURE 1.

Presented as a *New Instrument* at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

NEW INSTRUMENTS

OSCAR B. NUGENT, M.D.
CHICAGO, ILL.

ACRYLIC LENS APPLICATOR

The acrylic lens applicator (fig. 1) is a vacuum cup covered with neoprene rubber. The acrylic lens is held fast with its entire edge free, thus enabling the operator to place the lens behind the iris through the pupil without losing hold of it.

FIGURE 1.

SCLERA-CORNEAL SCISSORS

The sclera-corneal scissors (fig. 2) are made with short blades and longer handles. The short blades make it possible to cut the sclera or cornea without using side finger pressure and without danger of buckling of the tissues.

FIGURE 2.

ANTERIOR CHAMBER IRRIGATOR

The anterior chamber irrigator (fig. 3) is made of two luer-lok needles; one 22 gauge needle to which is attached a 5 cc. luer-lok syringe containing irrigating solution and one 16 gauge needle to which is attached an aspirating tube. While aspiration is being used the irrigating solution is gently forced into the anterior chamber, thus mixing it with the material being aspirated.

FIGURE 3.

Presented as *New Instruments* at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

NEW CAPSULOTOMY SCISSORS

JOHN M. MCLEAN, M.D.
NEW YORK, N. Y.

BECAUSE of the size of incisions necessary, the springing of incisions by scissor blades, and the serious risk of vitreous loss in the usual procedures of capsulotomy and iridocapsulotomy, it seemed worth while to try to devise capsulotomy scissors which would require a very small incision, preferably one which would be practically completely plugged by the scissors shaft. It also seemed advisable to design the scissors after the fashion of endoscopy instruments so that there would be no stress on the wound edges as the blades were opened and closed. With these features in mind the scissors shown in figure 1 were made. A small incision no larger than the shaft allows them to be introduced and there is practically no risk of vitreous loss as the opening is tightly plugged by the shaft. At the same time the blades may be opened and closed at will by pressing the button on the end of the shaft and manipulations may be made deliberately and without haste. The setscrew allows the blades to be set at any angle of rotation and at varying lengths of blade opening. This model has proved in practice to make the cutting of tough membranes a safer, simpler and more satisfactory procedure.

FIGURE 1.

From the Department of Surgery (Ophthalmology) of the New York Hospital and Cornell University Medical College.

Presented as a *New Instrument* at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

"BED-SPECS"

CLYDE H. JACOBS, M.D.
DANVILLE, PA.

FIGURE 1.

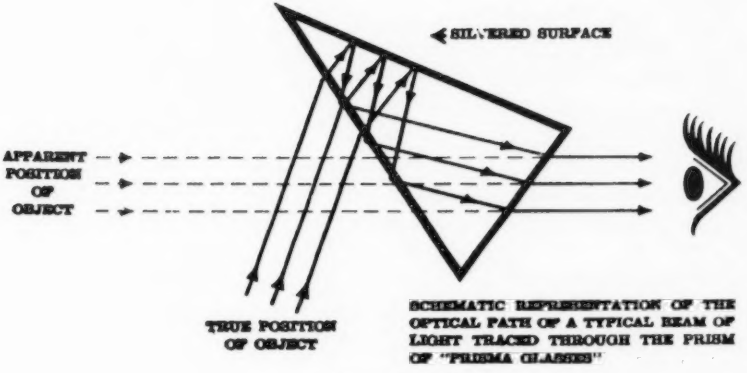

SILVERED SURFACE
APPARENT POSITION OF OBJECT
TRUE POSITION OF OBJECT
SCHEMATIC REPRESENTATION OF THE OPTICAL PATH OF A TYPICAL BEAM OF LIGHT TRACED THROUGH THE PRISM OF "PRISMA GLASSES"

FIGURE 2.

"BED-SPECS" were originally devised to be worn in a reclining position by bedfast patients, so that they might read in a supine position a book placed upon the upper abdomen (figs. 1 and 2). They also may be used for watching television.

With the prisms reversed, a person with the orthopedic entity known as

From the George F. Geisinger Memorial Hospital and the Foss Clinic.

Presented as a *New Instrument* at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

Strümpell-Marie disease can gaze straight ahead despite the severe angulation of the head downward (fig. 3). In conjunction with our Department of Orthopedics, we have used this adjunct successfully in numerous cases of this disease, as well as for a number of bedfast patients who wished to read.

FIGURE 3.

RHINOPHYMA: SURGICAL EXCISION FOLLOWING OBLIQUE CUTANEOUS INCISION

GERSON LOWENTHAL, M.D.
CINCINNATI, OHIO

RHINOPHYMA is a sequel to severe acne rosacea and requires surgical removal when the lesion becomes unsightly or when it obstructs breathing. Several methods of correction have been used. The commonest method for early cases involves shaving of the affected cutaneous areas of the lower third of the nose with a sharp knife or razor, followed by the application of a petrolatum dressing. In severe cases, plastic correction is performed by undermining and completely resecting the diseased area, followed by a full thickness or split thickness skin graft. Other reported methods include electrosurgical decortication alone, or with excision of the tumorous masses by divulsion followed by free skin grafting. The technic here reported is a modification of the shaving operation which has been successful in an advanced case of rhinophyma (fig. 1 A, B).

Following local anesthesia with procaine and epinephrine, the extent of the rhinophyma was outlined with methylene blue (fig. 2 A, B). An oblique cutaneous incision was made with a no. 11 blade along this outline, through the thickened hypertrophied epidermis extending to the corium. This incision was made at an angle to the vertical plane, forming a smooth gradation between the healthy skin and the derma underlying the lesion (fig. 2 C, D). By making the incision in this manner, a demarcation at the edge of the normal skin was avoided, and the cosmetic result was improved.

The lesion was pared away in layers with a sharp knife, care being taken to avoid damage to the alar cartilages. The excised tissues came away cleanly due to the angular incision, and were not prone to tear into the adjacent skin. By careful retraction of the lobulated masses around the nostrils during the excision, the normal vestibular border of the nares could be saved and subsequent notching prevented (fig. 2 D).

Following excision and shaping of the soft tissues of the nose, an absorbable gelatin sponge, saturated with topical thrombin, was applied over the wound to control oozing. A rubber tube, covered with petrolatum gauze, was inserted into each nasal fossa to maintain the nasal airway. A pressure dressing of fluffed gauze was applied externally over the saturated gelatin covering.

Parenteral penicillin was given to reduce secondary infection. The dressing was changed at weekly intervals. Crusts were removed, 10 per cent silver nitrate was applied to granulations, and a boric ointment dressing was used on the nineteenth postoperative day. The wound was healed in one month (fig. 1 C, D).

From the Department of Otolaryngology, College of Medicine, University of Cincinnati, and the Department of Otolaryngology, Jewish Hospital, Cincinnati, Ohio.

Presented as a *New Technic* at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

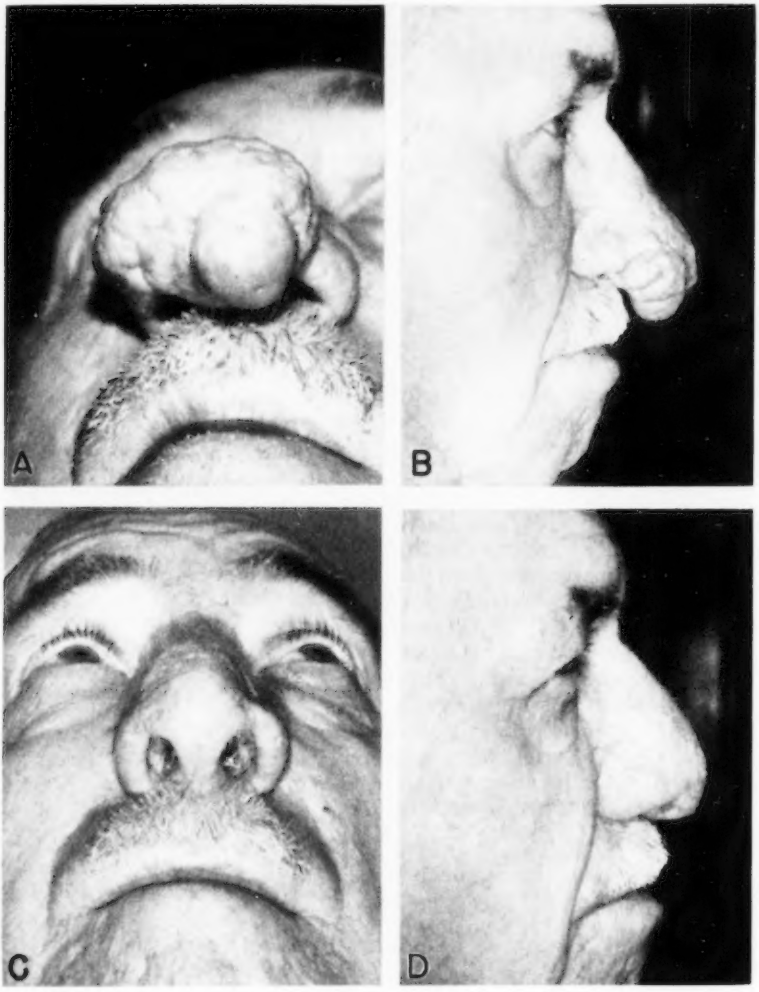


FIGURE 1.

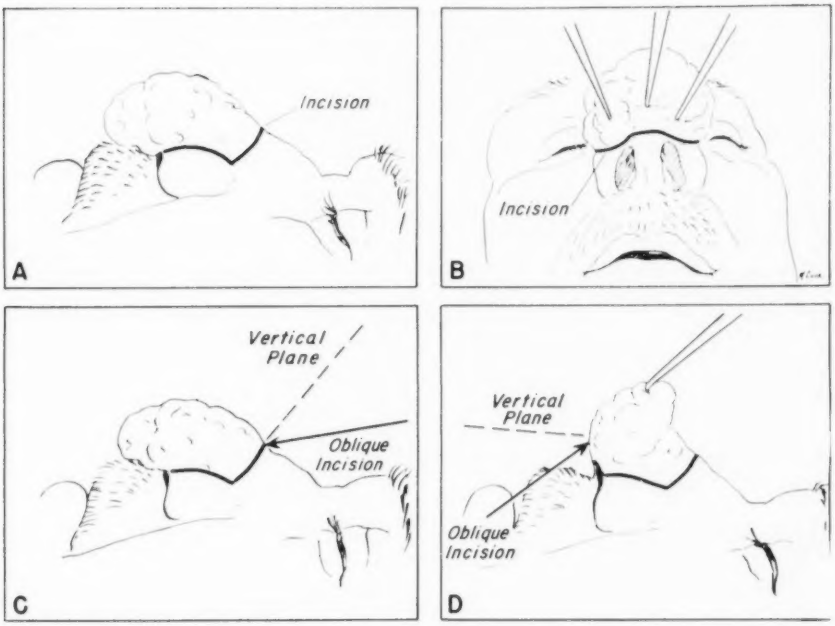


FIGURE 2.

This technic was employed in a severe case of rhinophyma, as shown in the figures. Thus a more extensive and complicated procedure involving the use of full thickness or split thickness skin grafts was obviated. A cosmetic result comparable to the other more complicated technics was obtained.

DIRECT VISION BONE CUTTING FORCEPS FOR RHINOPLASTY

IRVING B. GOLDMAN, M.D.
NEW YORK, N. Y.

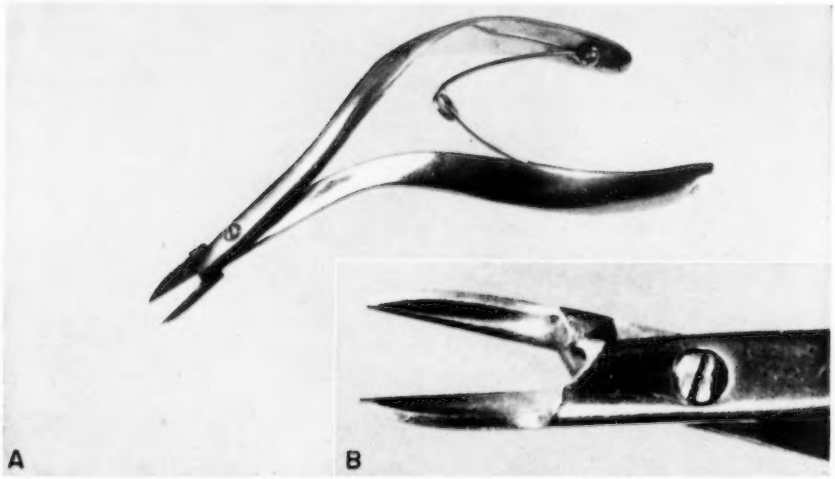


FIG. 1—A. Angular bone cutting forceps. B. Magnification of biting jaw showing contour of the cutting blades.

AFTER the bony hump has been removed the nasal processes of the maxillae are displaced mesially to obliterate the gap between the nasal bones. One not infrequently finds that on approximation the nasal bones are asymmetrical or that they project beyond the desired profile line producing a new pseudohump. Attempts to rasp or saw this projecting bone on an unstable pyramid is not only difficult but is fraught with danger. Scissors are not easily maneuvered nor have they the required power to amputate the desired fragment of bone accurately.

Based on the Kazanjian I have devised a bone cutting forceps (fig. 1) with nipper-type cutting blades for removal of fragments of the nasal bones or of any desired part of the bony vault under direct vision. Both cutting blades are in a slanted position towards the cutting edge which affords observation for the precise amount of bone to be removed. The instrument has a bent handle permitting a clear and direct view of the field of operation.

From the Division of Rhinoplasty, Department of Otolaryngology, Mount Sinai Hospital, New York.

Presented as a *New Instrument* at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

Manufactured by Klenk Company, Maspeth, L. I., N. Y.

A NEW SET OF ADENOID CURETTES

EDLEY H. JONES, M.D.
VICKSBURG, MASS.

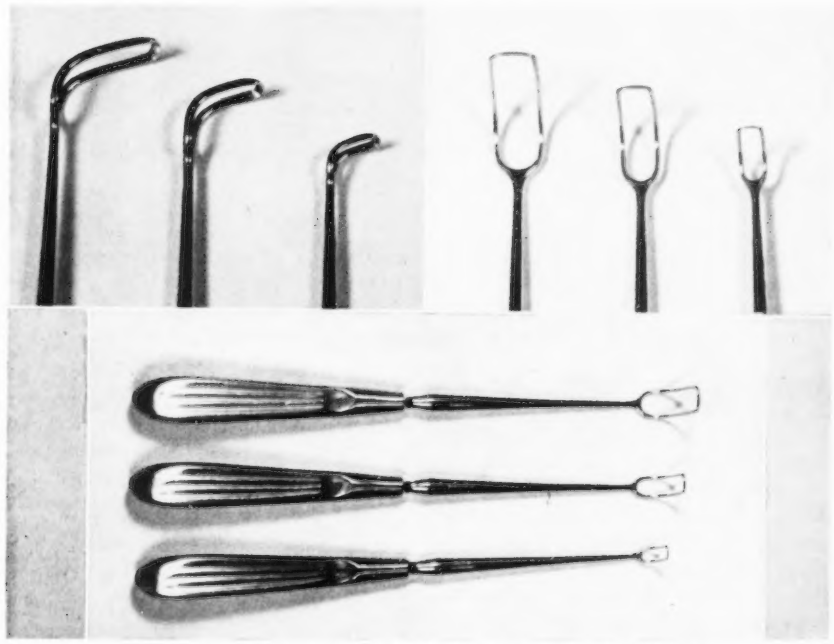
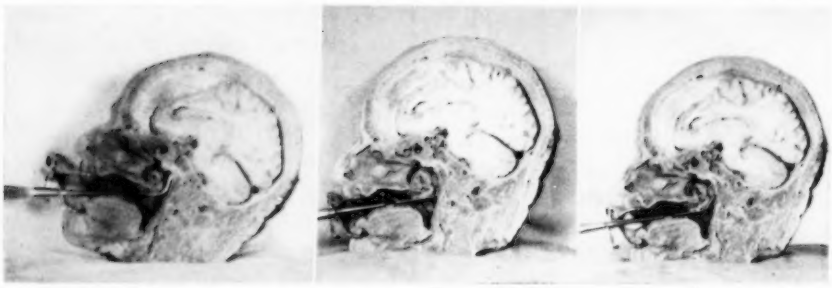


FIG. 1.—Views of new curettes, showing details of size, height and angle.

ADENOIDECTOMY is generally considered the most poorly performed operation. Nasopharyngoscopic examination after adenoidectomy all too frequently reveals an amazingly large amount of adenoid tissue left in situ. Such poor results have attracted the attention of many otolaryngologists who have attempted to improve the procedure. One of these, Dr. Henry E. Middleton, designed and described the Rosenmüller fossa curette before the Illinois State Medical Association in 1935, though he never described it in the literature. In response to my inquiry he wrote, "I remove the larger mass of adenoid tissue with the LaForce Adenotome and finish with the Rosenmüller fossa curette. I hold this much as one holds a pen and bring the cutting blade to bear with the proper pressure, giving the curette a sweeping motion behind the eustachian

Presented as a *New Instrument* at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.



A B C

FIG. 2—Comparison of the angle and height of (A) the new curette with (B) the Vogel curette and (C) the Rosenmüller fossa curette.

cushion. I am always able to remove considerable additional tissue." It is my pleasure to give him full credit for designing this valuable instrument.

However, with this instrument, and all other available curettes, retractors and punches the author still noted embarrassing amounts of adenoid tissue remaining postoperatively, usually clumped around the posterior attachment of the nasal septum or mushrooming upward into the posterior choanae; and determined to design an instrument that could remove such tissue.

After obtaining measurements of the dimensions of the posterior choanae of a group of skulls of children and adults of various ages (none were available in the literature) and studies of the angle of the nasopharynx and pharynx in anatomical specimens, the author designed a modification of the Middleton curette, with a different angle and of three sizes (fig. 1). The change in angle and length of jaws permits the curette to be introduced into the posterior choanae (fig. 2). The adult size has an outside width of 10 mm. with a length of 20 mm.; the child size is 8 by 15 mm. and the infant size is 6 by 10 mm., as illustrated. The infant size will enter the posterior choanae of a 2-year-old infant. The child size is the one most often used.

The technic of usage is to place the curette high in the posterior choanae with the jaws and blade flat against the posterior wall, then remove the adenoid tissue by shaving straight down the wall. The blade should be razor-sharp. Anatomical studies revealed that many nasopharynges slope posteriorly and laterally, but if this technic is followed the adenoid tissue can be cleanly removed from around the posterior attachment of the nasal septum, the posterior choanae and around Rosenmüller's fossa, without damaging the underlying tissues.

The author wishes to express his appreciation to Dr. Marc P. Moldawer (anthropologist) for measurements of skulls from the Department of Anatomy, Harvard University; to Dr. Harold Cummins, Professor of Anatomy at Tulane University, for similar measurements and for the privilege of studying anatomical specimens at the Tulane Department of Anatomy; and to V. Mueller and Company for their cooperation in designing the instruments.

A NEW LARYNGEAL DILATOR

EDLEY H. JONES, M.D.
VICKSBURG, MISS.

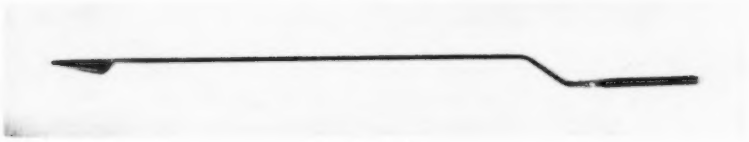


FIGURE 1.

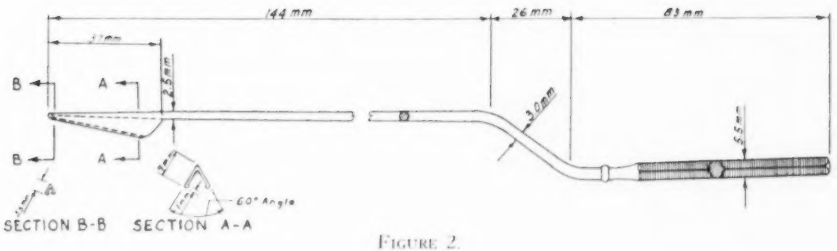


FIGURE 2.

FOR children who require laryngeal dilation under ether anesthesia, it is sometimes desirable to leave the dilator in place for a few minutes. Obviously this cannot be done with existing models, such as the Jackson triangular laryngeal dilator.

To meet this need, a V-shaped dilator was devised with over-all dimensions that could be passed through a 12 cm. Jackson laryngoscope, the tip being small enough to enter an infant's larynx, the flanges increasing to full size at the superior margins.

This instrument was designed for use in children but a larger size can be made for adults (figs. 1 and 2).

This instrument can be inserted and left in situ as long as desired, as it provides a clear airway.

Manufactured by George P. Pilling and Son Company. The author wishes to thank Mr. Henry Pilling for his assistance.

Presented as a *New Instrument* at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

TRACHEOTOMY TUBE FOR INHALATION THERAPY

ALBERT H. ANDREWS, JR., M.D.
CHICAGO, ILL.

THE APPLICATION of inhalation therapy to tracheotomy is in some instances of extreme value. Oxygen, helium and oxygen, carbon dioxide, water vapor, therapeutic aerosols, and positive pressure are procedures which may be used. The attachment to the tracheotomy tube should not add any obstruction to air flow, set up extreme eddy currents, or interfere with aspiration through the tracheotomy tube, and should not require interruption of the gas therapy when aspiration is done.

A laryngectomy tube fulfilling these criteria has been described[1] and similar tracheotomy tubes have been developed.* The standard Jackson tracheotomy tube with rotating collar has an additional inner tube with an extension and side arm. A metal cap seals one tube and is removed when aspiration is done. A chain fixes the cap to the tube so that it is controlled at all times.

The 00 and 0 tracheotomy tubes are supplied without the side tube because the inner tube is so small that aspiration must be done with the inner tube removed.

The O.E.M. mask for tracheotomy may be used. An oxygen humidifier of the type used for oxygen catheter therapy will furnish a moderately high humidity. When air dilution is employed by means of the special metering device, a steam inhaler is placed so as to deliver the steam at the air intake of the meter.

When gas therapy is not desired, the special inner tube is removed and the conventional one inserted.

* Produced by the George P. Pilling and Son Company, Philadelphia, Pa.

From the Children's Memorial and St. Luke's Hospitals, Chicago.

Presented as a *New Instrument* at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

REFERENCE

1. Andrews, A. H., Jr.: Laryngectomy tube for oxygen therapy, Laryngoscope, 62:752-753 (July) 1952.

TRANSACTIONS
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Official Publication of the American Academy of Ophthalmology and Otolaryngology
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HENRY L. WILLIAMS, Rochester, Minnesota, *Associate Editor*
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All communications regarding the TRANSACTIONS should be addressed to William L. Benedict, M.D., Executive Secretary-Treasurer, American Academy of Ophthalmology and Otolaryngology, 100 First Avenue Building, Rochester, Minnesota

Editorials

*Supplementary Report of the Board of Trustees Concerning the Resolution on Definition of Medical and Dental Services.** Following a meeting with the American Board of Oral Surgery (Dental), the American Dental Association, and the American Hospital Association, the agreed recommendation was as follows:

The position adopted by the representatives at the meeting on March 21 was to the effect that the oral (dental) surgeon be assigned to the surgical service of a hospital and to perform such professional duties as the chief of the surgical service directed. The members of the American Board of Oral (Dental) Surgery stated that this plan was one which they drafted, similar to that of the Joint Commission on Accreditation of Hospitals, and was fully satisfactory.

Your reference committee recommends that the word "the" preceding the phrase "surgical service of a hospital" be changed to the word "a" so that it would then read, "be assigned to a surgical service in a hospital."

Your reference committee further recommends that the following definition of a dental-oral surgeon be included in the report:

Dental-oral surgery is to be limited to diseases of the teeth and jaws and lesions of contiguous soft tissues related to diseases of the teeth and jaws but excluding malignancies.

*Proceedings of the New York Meeting, J.A.M.A., 152:731 (June 20) 1953.

Letter to the Editor

Dear Doctor Benedict:

As you know, we now have five prints of your film entitled EMBRYOLOGY OF THE EYE. Recently, we analyzed bookings on all films in our library for the 10-month period beginning September 1, 1952. We find that the five prints of EMBRYOLOGY OF THE EYE were booked more than sixty times and it ranks among our six most popular pictures.

Anticipating a greater demand for all of our films in 1953, I have decided to order an additional print.

RALPH P. CREER, Secretary

Committee on Medical Motion Pictures
American Medical Association

In Memoriam

Henry Bacon Abbott.....Geneva, New York
July 23, 1953

Herbert Oscar Brust.....Syracuse, New York
April 30, 1953

Richard Edison Estlick.....Fort Wayne, Indiana
April 6, 1953

Morris Arthur Glatt.....Beverly Hills, California
July 19, 1953

Lewis Pellman Glover.....Altoona, Pennsylvania
April 22, 1953

Paul Bloch Grogin.....Pittsburgh, Pennsylvania

Frederick Maximilian Grossman.....Seattle, Washington
May 12, 1953

Mary Kathrina Heard.....St. Petersburg, Florida
April 20, 1953

David Morrison Johnson.....Columbus, Ohio
May 14, 1953

Marvin Luther Latimer.....New Haven, Connecticut
April 17, 1953

Roy Seeley Moore.....Syracuse, New York
May 26, 1953

John Gillis Sanders.....Foley, Alabama
June 1, 1953

Victor Conway Smith.....New Orleans, Louisiana
May 19, 1953

George William Stimson.....Warren, Ohio
April 20, 1953

FIFTY-EIGHTH ANNUAL MEETING

American Academy of Ophthalmology and Otolaryngology

OCTOBER 11 TO 16, 1953

PALMER HOUSE

Chicago, Illinois

OFFICIAL NOTICES

Convention Call

Under the authority of the Constitution and with the approval of the Council, I herewith proclaim that the Fifty-Eighth Annual Session of the American Academy of Ophthalmology and Otolaryngology will convene at the Palmer House, Chicago, Illinois, on Monday, October 12, 1953 at 9:30 a.m., and will continue through noon of Friday, October 16, 1953.

FREDERICK C. CORDES, M.D.
President

Attest:
WILLIAM L. BENEDICT, M.D.
Executive Secretary-Treasurer

Ladies' Committee

Mrs. Gail R. Soper and
Mrs. Roland D. Russell, *Co-Chairmen*
Mrs. Arthur J. Coombs
Mrs. Ralph Davis
Mrs. Robert Fitzgerald
Mrs. Earle B. Fowler
Mrs. Stanton A. Friedberg
Mrs. Richard Gamble
Mrs. Elmer W. Hagens
Mrs. William Hughes
Mrs. Peter Kronfeld
Mrs. Lawrence J. Lawson
Mrs. Francis L. Lederer
Mrs. John R. Lindsay
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Mrs. Samuel J. Meyer
Mrs. Henry G. Mundt
Mrs. Frank Newell
Mrs. Samuel J. Pearlman
Mrs. Karl J. Scheribel
Mrs. Sherman L. Shapiro
Mrs. William A. Smiley
Mrs. Derrick Vail
Mrs. Oliver E. Van Alyea

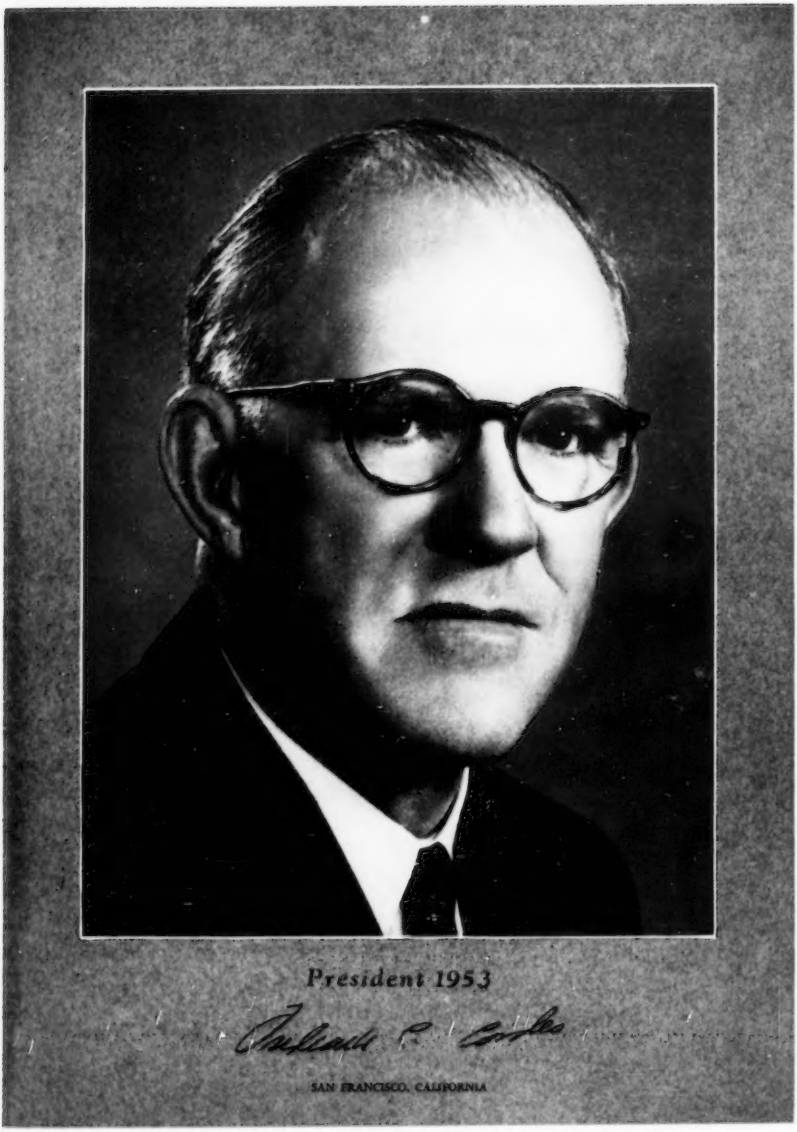
Business Meeting

The Annual Business Meeting of the Academy will be held in the Crystal Room of the Palmer House on Thursday, October 15, 1953, at 5:30 p.m.

The Order of Business:

1. Call to order
2. Report of Executive Secretary-Treasurer
3. Report of the Senior Member of the Council
4. Election of Fellows (See Candidate List, p. 644)
5. Election of Officers
6. Unfinished business
7. New business

All Fellows and candidates for election to Fellowship are urged to attend.



President 1953

SAN FRANCISCO, CALIFORNIA

Guest of Honor

LAWRENCE T. POST

Graduate of the Johns Hopkins University School of Medicine, 1913. Certified by the American Board of Ophthalmology, 1920. Emeritus Professor of Clinical Ophthalmology and Head of the Department of Ophthalmology, Washington University School of Medicine. Member of the Subcommittee on Ophthalmology, National Research Council. Life Fellow of the American Academy of Ophthalmology and Otolaryngology, and President in 1944. Fellow of the American College of Surgeons. Member of the American Ophthalmological Society and of the Association for Research in Ophthalmology. Awarded Leslie Dana Medal for outstanding achievement in the prevention of blindness, 1948.

Council Meetings

The Council of the American Academy of Ophthalmology and Otolaryngology will convene at 9 o'clock, Saturday morning, October 10, 1953, in Private Dining Room 4 on the third floor of the Palmer House, Chicago. There will also be a meeting of the Council on Wednesday, October 14, at 12:30 in Private Dining Room 5. Members desiring to be heard or to present business will confer with the Executive Secretary-Treasurer for appointments.

FREDERICK C. CORDES, M.D.
President

New Business

Proposals for new activities often are presented too late during the Academy session to afford time for deliberation. Therefore, new items requiring Council action should be presented in writing to the Executive Secretary-Treasurer early so that the Committee concerned may have an opportunity to study the matter prior to presentation to the Council.

Public Relations

Contributors to the scientific program are requested to send copies of their papers to the Secretary for Public Relations, Erling W. Hansen, M.D., 90 South Ninth St., Minneapolis 2, Minn., at the earliest possible moment.

Exhibits

The Scientific and Technical Exhibits will open at 9 o'clock Sunday morning, October 11. They will close at 4 o'clock Sunday for the President's Reception. From Monday through Friday they will open at 8:30 a.m. and close at 6:00 p.m. except that they will close Friday, October 16, at 12 o'clock.

Dues

Receipt card for 1953 dues is required for registration at the Academy meeting. Those who have not paid dues for 1953 are requested to remit to W. L. Benedict, M.D., Executive Secretary-Treasurer, 100 First Avenue Bldg., Rochester, Minn. In case of loss, a duplicate receipt card may be obtained upon request.

Registration

General registration will begin at 9 o'clock Sunday morning, October 11, in the Foyer to the Grand Ball Room, Fourth Floor.

The regulation badge secured at the time of registration is essential for admittance to all activities. To facilitate registration the following sections will be provided:

Advance Orders: Members who have reserved instruction course tickets register here. Advance orders are held here for collection.

Members Without Advance Orders: Please present Receipt Card for 1953 dues. Dues outstanding may be paid at this section.

(Application blanks for Fellowship in the Academy may be obtained here.)

Instructors: All persons presenting instruction courses register here to secure special badge. Instructors who are not members of the Academy may register without fee.

Nonmembers: A registration fee of $5.00 is required of all guests (see next page). The following exceptions are made:

1. Candidates for Academy Fellowship whose names appear in the July-August issue of the TRANSACTIONS.
2. Residents in training and registrants for the 1953-1954 Home

Study Courses presenting guest cards obtained before the meeting (see below).

3. Guests taking part in the Scientific Program or Exhibits.

Orthoptic Technicians: All orthoptic technicians register here. Members of the American Association of Orthoptic Technicians who have ordered tickets for Orthoptic Technicians Instruction Courses may pick them up here. Tickets will be on sale for those who have not yet purchased them.

Guests

Physicians who are not members are welcome to attend the meeting of the Academy as guests and to participate in all the activities. All guests, however, will be required to register and a fee of $5.00 will be charged.

In the past, a number of Fellows and registered guests of the Academy have indicated that they wish their *office nurses, technicians,* and *similar personnel* to attend parts of the Academy program or exhibits. Such individuals will be allowed to register upon presentation of a written request from a Fellow or if accompanied by a registered guest or Fellow. The guest fee of $5.00 will be required. An exception is made for all members of the American Association of Orthoptic Technicians, who may register without fee.

Residents and 1953-1954 Registrants for Home Study Courses

Men in training in accepted residencies, or those now completing a full academic year (nine months) in basic study, may register without fee upon presentation of a guest card obtained by written request of their Department Heads.

Registrants who have completed enrollment for the 1953-1954 Home Study Courses in Ophthalmology or Otolaryngology may also request a guest card entitling them to registration without fee.

Requests from Department Heads and Home Study Courses registrants for guest cards should be sent to W. L. Benedict, M.D., 100 First Avenue Bldg., Rochester, Minn., by October 1.

Instruction Course Tickets

The tickets for courses in ophthalmology and the tickets for courses in otolaryngology and maxillofacial surgery will be sold at separate desks in the Registration Headquarters. The bulletin boards behind each desk will indicate what tickets are available. Please consult these boards in making your selections. Bear in mind that individual courses are for one hour only; continuous courses run for two or more hours. Price of tickets is $2.00 per hour.

It is necessary to register and to be wearing regulation badge in order to purchase instruction course tickets.

Ladies' Registration

The wives of members and guests are urged to register at the Ladies' Headquarters in Rooms 15 and 16 on the Club Floor. Registration will open at 9 o'clock, Sunday morning, October 11, and will close at noon on Thursday, October 15.

Available Supplies

Collected abstracts of instruction courses, manuals and monographs may be purchased at the supply counter in Registration Headquarters. Guests may place subscriptions to the bimonthly issues of the TRANSACTIONS here, and orders will be taken for the 1953 Bound Volume.

Dinner Tickets

Tickets for Alumni Dinners will be on sale at the Registration Headquarters until 2 o'clock Monday afternoon.

Tickets for the Annual Banquet of the Academy, which will be held on Wednesday evening, may be purchased at Registration Headquarters before 2 o'clock on Wednesday afternoon. The price will be $6.00.

Constitutional Changes in Fellowship Status

Life Fellows. Fellows who have paid dues continuously for thirty years will automatically become Life Fellows. They shall not be required to pay any fees or dues or assessments and shall enjoy all the privileges of Active Fellows.

Senior Fellows. Fellows who have paid dues continuously for twenty-five years will be termed Senior Fellows and their dues will be one-half of the dues of Junior Fellows.

Article V, Section 6, By-Laws

Life Fellows — 1954

Ballon, David H. ...Montreal, Que., Canada

Chase, W. D. .....Bethlehem, Pa.
Croman, Joseph M., Jr. ..Mt. Clemens, Mich.
Cushman, Beulah .....Chicago, Ill.

Davidson, H. P. .....Chicago, Ill.
Dunnington, John H. .....New York, N. Y.

Flynn, James A. .....Washington, D. C.

Glasford, S. T. .....Pekin, Ill.
Goodyear, Henry Marks ....Cincinnati, Ohio
Gracey, George F. .....Harrisburg, Pa.

Hall, Alice K. .....Oak Park, Ill.
Heath, Parker .....Winchester, Mass.
Hogan, W. L. .....Hartford, Conn.

Joynt, Albert J. .....Waterloo, Iowa

Kilbourne, P. H. .....Dayton, Ohio
Kirby, Daniel B. .....New York, N. Y.

Morton, J. B. .....Detroit, Mich.
Pearlman, S. J. .....Chicago, Ill.
Reuling, F. H. .....Waterloo, Iowa
Rice, John E. .....Worcester, Mass.
Richards, Lyman G. .....Brookline, Mass.
Russell, Ralph Emerson ....Waterloo, Iowa

Schuster, S. A. .....El Paso, Texas

Senior Fellows — 1954

Adkins, George E. .....Jackson, Miss.
Adler, Francis Heed .....Philadelphia, Pa.
Anthony, D. Harbert .....Memphis, Tenn.
Aronson, Sidney S. .....Indianapolis, Ind.
Atkinson, Thomas E. .....Greeley, Colo.

Baeseman, R. Winfield .....Allenhurst, N.J.
Barkan, Otto .....San Francisco, Calif.
Barnett, Saul E. .....Detroit, Mich.
Beideman, Joseph E. .....Norristown, Pa.
Bolotow, Nathan A. .....Providence, R. I.
Brawner, Leon E. .....St. Simon Island, Ga.
Bredlau, Ernest A. .....Chicago, Ill.
Briant, Thomas E. ....Welland, Ont., Canada

Cardwell, Edgar P. .....Newark, N. J.
Carter, John M. .....Detroit, Mich.
Cave, Linus S. .....Syracuse, N. Y.
Childers, Robert J. .....Plainfield, N. J.
Cone, Alfred J. .....St. Louis, Mo.
Crane, Walter R. .....Los Angeles, Calif.

Davidson, Morris M. .....Jackson, Miss.
Dixon, Fred W. .....Cleveland, Ohio
Duncan, Robert A. .....Amarillo, Texas

Fraser, Robert C. .....Los Angeles, Calif.

Gamble, Richard C. .....Chicago, Ill.
Good, William M. .....Waterbury, Conn.
Gordon, Glenn G. .....Baker, Ore.
Grecar, James N., Jr. .....Reno, Nev.
Griffey, Edward W. .....Houston, Texas
Gunn, Milus L. .....Harlan, Ky.
Guttman, M. Reese .....Chicago, Ill.

Haessler, F. Herbert .....Milwaukee, Wis.
Hanson, William L. .....East St. Louis, Ill.
Hobart, Carl .....St. Louis, Mo.
Hofling, Charles A. .....Cincinnati, Ohio
Hoople, Gordon D. .....Syracuse, N. Y.
Hughes, Wendell L. ..Hempstead, L. I., N. Y.

Keefe, William J. .....Mt. Vernon, N. Y.
King, George L. .....Alliance, Ohio
Kleinert, Margaret N. .....Boston, Mass.
Koebbe, Ezra E. .....Columbus, Neb.

Lamb, Harry H. .....Davenport, Iowa
Le Win, Thurber .....Buffalo, N. Y.

Marks, Julian B. .....Los Angeles, Calif.
Martin, Roy D. .....Grand Island, Neb.
Mayerberg, Emil R. .....Wilmington, Del.
McCullough, Clarence J. ...Washington, Pa.
McDavitt, Bertha S. S. .....Temple, Texas
McGarvey, William E. .....Jackson, Mich.
Motto, M. Paul .....Cleveland, Ohio

Nash, C. Stewart .....Rochester, N. Y.
Nemours, Paul R. .....Seattle, Wash.

Pelz, Mort D. .....St. Louis, Mo.
Pinkerton, Forrest J. .....Honolulu, Hawaii
Potter, Allen B. .....St Louis, Mo.

Richardson, James J. ....Fort Worth, Texas
Robb, Mayo .....Phoenix, Ariz.
Romonek, Philip L.....Beverly Hills, Calif.
Rosenberger, Harry C. .....Cleveland, Ohio
Rucker, C. Wilbur .....Rochester, Minn.
Rudolphy, Jay B. .....Philadelphia, Pa.
Ruedemann, A. D., Sr. .....Detroit, Mich.

Samuels, Bernard .....New York, N. Y.
Schwartz, Virgil J. .....Minneapolis, Minn.
Shuster, Benjamin H. .....Philadelphia, Pa.
Sidwell, Clarence E. .....Longmont, Colo.
Simpson, John R. .....Huntingdon, Pa.
Smith, Carroll L. .....Spokane, Wash.
Smith, Ruric N. .....Tulsa, Okla.
Spiesman, Irwin G. .....Maywood, Ill.
Sutherland, J. M. .....Detroit, Mich.

Tassman, Isaac S. .....Philadelphia, Pa.
Theobald, Georgiana Dvorak ..Oak Park, Ill.
Theobald, John J. .....Oak Park, Ill.

Warner, Robert J. .....Nashville, Tenn.
Woodward, Fletcher D. ..Charlottesville, Va.

LOCATION OF ROOMS
SCHEDULED FOR ACADEMY ACTIVITIES

THIRD FLOOR	FOURTH FLOOR	CLUB FLOOR
P.D.R. 1 through 9	Exhibit Hall	P.D.R. 14 through 18
P.D.R. 11	Red Lacquer Room	
Crystal Room	Foyer to Grand Ball Room	
	Grand Ball Room	
	Executive Office	
	Public Relations Office	

Palmer House
Chicago

P.D.R.—Private Dining Room

Club Floor may be reached by stairway from Fourth Floor

CONVENTION ACTIVITIES

PRESIDENT'S RECEPTION

Grand Ball Room
Sunday, 4:30 p.m.

President Frederick C. Cordes and Mrs. Cordes invite all members and guests of the Academy to a reception honoring Dr. and Mrs. Lawrence T. Post, Mr. Frank Law of London, England, and Dr. and Mrs. Thomas C. Galloway on Sunday, October 11, at 4:30 p.m. in the Grand Ball Room.

JACKSON MEMORIAL LECTURE

Sponsored by the Ophthalmic Publishing Co.

Dr. Harold F. Falls, Professor of Ophthalmology at the University of Michigan Medical School, will deliver the Jackson Memorial Lecture at the scientific session of the Section on Ophthalmology, Thursday morning, October 15. The subject of his paper will be "Clinical Detection of the Carrier State in Ophthalmologic Pathology."

WHERRY MEMORIAL LECTURE

Sponsored by the Wherry Memorial Fund

The 1953 Wherry Memorial Lecture will be delivered at the scientific session of the Section on Otolaryngology, Thursday afternoon, October 15, by Dr. George E. Shambaugh, Jr., Department of Otolaryngology of Northwestern University Medical School. The subject of his address will be "Some Contributions of Laboratory Research to the Development of Fenestration Surgery."

EVENING OF DIVERSION

Grand Ball Room
Monday, 9:00 p.m.

Entertainment, refreshments and music for dancing. Complimentary.

ANNUAL ACADEMY DINNER

Grand Ball Room
Wednesday, 7:00 p.m.

Dr. Robert Gordon Sproul, President of the University of California, will be the dinner speaker. Dr. Sproul in his activities as President of the University has always been vitally concerned with the development and advancement of medicine and medical education. He is a director of the American Foundation of Tropical Medicine, a member of the National Red Cross Advisory Committee, Special Consultant to the Office of Scientific Research and Development, and a member of the Executive Council of the American Cancer Society.

Dr. Sproul is also intensely interested in international affairs, especially Pan-American activities. He is a trustee of the Institute of International Education, of the World Affairs Council, of the Carnegie Foundation for Advanced Teaching, and of the Rockefeller Foundation, as well as Senior Advisor of the American Group on Allied Committee on Reparations.

FACULTY LUNCHEON

Private Dining Room 14
Thursday, 12:45 p.m.

President Frederick C. Cordes will be host to all those presenting scientific papers, motion pictures, scientific exhibits, and courses of instruction.

ALUMNI DINNERS

The following alumni groups will hold dinners on Monday evening, October 12, 1953, at 6:30 p.m., unless time is otherwise indicated: (A complete listing including place of dinner and address of the secretary for each group will appear in the Pocket Program issued at the Academy meeting.)

Barnhill-McCaskey Alumni
Boston City Hospital Aural & Ophthalmic Society
Brooklyn Eye and Ear Alumni Association
Dr. J. Mackenzie Brown Alumni
University of Colorado Alumni
Cook County Hospital Alumni
Emory University Alumni (Former Staff Members)
Henry Ford Hospital Alumni—6:00 p.m.
Gill Memorial Eye, Ear and Throat Hospital Alumni—Annual Spring Graduate Course
Harper Hospital Alumni
Illinois Eye and Ear Infirmary Alumni
Iowa Eye, Ear, Nose and Throat Alumni—Cocktails 5 p.m.
Iowa Ear, Nose and Throat Alumni
Dr. Chevalier Jackson Alumni
Jefferson Hospital Otolaryngological Group Alumni
Johns Hopkins Hospital—Wilmer Institute and Otolaryngology Alumni
Dr. John R. Lindsay Alumni
Manhattan Eye, Ear and Throat Hospital—Society of Ex-House Surgeons
Massachusetts Eye and Ear Infirmary Alumni—Ophthalmology
Massachusetts Eye and Ear Infirmary and Harvard Medical School Alumni—Otolaryngology
Mayo Clinic Alumni
Mount Sinai Hospital of New York Alumni
University of Nebraska College of Medicine Alumni
New Orleans E.E.N.T. Hospital—Charity Hospital Alumni
New York Eye and Ear Infirmary Alumni
New York University, New York Post Graduate Medical School and Bellevue Hospital Alumni
Otolaryngologic Alumni Association of the University of Illinois Eye and Ear Infirmary
Walter R. Parker-Roy Bishop Canfield Alumni
University of Pennsylvania and Wills Hospital Alumni—Ophthalmology
University of Pennsylvania Alumni—Otolaryngology
Presbyterian Hospital (New York) Ear, Nose and Throat Group
St. Luke's Hospital Alumni
Washington University Alumni—Ophthalmology
Washington University Alumni—Otolaryngology
Wilmer Institute Alumni—See Johns Hopkins Hospital

OUT-OF-PRINT ISSUES

The Academy office receives frequent requests for volumes of the TRANSACTIONS which are now out of print. If you have volumes before 1920 which you wish to dispose of, please notify Dr. W. L. Benedict, 100 First Avenue Building, Rochester, Minnesota.

CANDIDATES--1953

The following candidates have qualified for Fellowship in the American Academy of Ophthalmology and Otolaryngology and will be voted upon at the Annual Business Meeting, Thursday, October 15, 1953. Objections to the election of a candidate must be filed with the Executive Secretary-Treasurer previous to Saturday, October 10, 1953.

CANDIDATES CERTIFICATED BY BOARDS

Ophthalmology (OP)
Otolaryngology (ALR)
Plastic Surgery (Pl S)

Abernethy, Paul McBee, Box 758, Burlington, N. C., OP
Abernethy, Rodney Elmer, 12 N. El Camino Real, San Mateo, Calif., OP
Adams, Samuel T., 1610 Pine Ave. West, Montreal 25, Que., Canada, OP
Alfano, Joseph Edward, 122 S. Michigan Ave., Chicago 3, Ill., OP
Allen, Henry Freeman, 200 Beacon St., Boston 16, Mass., OP
Anderson, William Henley, Jr., Commercial Bank Bldg., Ocala, Fla., OP
Andrew, James M., 269 E. State St., Columbus 15, Ohio, OP
Ariagno, Richard P., 55 E. Washington St., Chicago 2, Ill., ALR
Arragg, Fred G., 301 Essex St., Lawrence, Mass., ALR
Arrington, Jerry E., 9 Buttles Ave., Columbus 8, Ohio, ALR
Arthur, John Edward, 514 W. Church Ave., Knoxville, Tenn., ALR
Ascher, Karl Wolfgang, 5 W. Fourth St., Cincinnati 2, Ohio, OP
Atkinson, Marshall Beale (Major), 3901 South Q St., Fort Smith, Ark., OP

Babb, John William, 330 Queen's Ave., London, Ont., Canada, ALR
Bach, Mark John, 324 E. Wisconsin Ave., Milwaukee 2, Wis., ALR
Baker, Philip Harriman, 307 N. Locust St., Inglewood 1, Calif., OP
Baldwin, James Lee, 816 Medical Arts Bldg., Dallas 1, Texas, ALP
Ballard, Joseph Aloysius, 203 W. Second St., Lexington 6, Ky., ALR
Banoff, Harry, U. S. Army Hospital, Fort Benning, Ga., OP
Baras, Irving, 3345 Medical Group, Chanute Air Force Base, Rantoul, Ill., OP
Barratt, William E., Mitchel Air Force Base, Hempstead, N. Y., ALR
Barrett, Earl L., 608-9 General Insurance Bldg., Seattle 5, Wash., OP
Bartlett, Isaac E., 213 W. Wesley St., Wheaton, Ill., ALR
Barton, Stanley L., 269 E. State St., Columbus 15, Ohio, OP
Baumgarten, Otto Charles, U. S. Naval Hospital, Corona, Calif., OP
Baxter, William Frank, 1624 Franklin St., Oakland 12, Calif., ALR
Bayard, Walter Locke, 900 Courtland, Park Ridge, Ill., OP
Beaton, Alexander H. (Capt.), 2114-1 U. S. Army Hospital, Camp Pickett, Va., ALR
Beaty, Harold W., 118 Colony St., Meriden, Conn., ALR
Bedrossian, Robert Haig, 4501 State Rd., Drexel Hill, Pa., OP
Bell, Nelson Clark, 1708 N. Garey Ave., Pomona, Calif., OP
Bennett, David, P. O. Box 9, Orange, Texas, ALR
Benton, Curtis D., Jr., 15 S. E. 16th St., Fort Lauderdale, Fla., OALR
Berg, Joseph L., 305 N. Jefferson St., Albany, Ga., OP
Bergendahl, Emil Henry, 629 Medical Center Bldg., Fort Wayne 2, Ind., ALR
Bergmann, Robert Bruce, 959 Bushwick Ave., Brooklyn 21, N. Y., OP
Berry, Frank D., P. O. Box 474, c/o O'Neill, Ojai, Calif., OP
Bickerton, John Harvey, Armed Forces Institute of Pathology, Washington 25, D. C., OP
Bishop, John Walter, 1162 Willamette St., Eugene, Ore., OP
Bixler, Donald P., 1410 Brown St., Anderson, Ind., OP
Blackhurst, Robert Thomas, Blackhurst Bldg., Midland, Mich., OP
Blender, William, 630 Jefferson Bldg., Peoria, Ill., OP
Booth, Lee Norton, 1138 Elm St., Manchester, N. H., OP

Bordley, John Earle, The Johns Hopkins Hospital, Baltimore 5, Md., ALR
Botsford, Daniel R., 3240 Main St., Buffalo 14, N. Y., OP
Bounds, George W., Jr., University Hospitals, Iowa City, Iowa, OP
Bowers, Berna Thomas, Fitzsimons Army Hospital, Denver 8, Colo., OP
Bradley, Wesley Holmes, 1100 E. Genesee St., Syracuse, N. Y., ALR
Brandt, John Philip, 216 E. Water St., Lock Haven, Pa., OP
Bresette, James Edward, 659 New Brotherhood Bldg., Kansas City, Kan., OP
Brizzolara, Aristo John, 408 Donaghey Bldg., Little Rock, Ark., ALR
Brody, Michael, 228 Livingston Ave., New Brunswick, N. J., OP
Brookshire, Paul Franklin, Jr. (Major), Madigan Army Hospital, Tacoma, Wash., ALR
Brown, Delatus Earl, 250 Fidelity Medical Bldg., Dayton 2, Ohio, OP
Brown, James W. (Col.), Ellsworth Air Force Base, Rapid City, S. D., OP
Brown, James Walter, Jr., 205 Cabarrus Bank Bldg., Kannapolis, N. C., ALR
Browning, Carroll Welles, 101 N. Zangs Blvd., Dallas 8, Texas, OP
Bryan, Burton Donald, 151 Rock St., Fall River, Mass., ALR
Buckhaults, Wendell Wallace, 103 E. Jones St., Savannah, Ga., OP
Buckingham, Richard Albert, 510 S. Cuyler St., Oak Park, Ill., ALR
Burell, Vincent A., 218 Chambers St., Phillipsburg, N. J., OP
Burnside, Ronald M., 4105 Live Oak St., Dallas 1, Texas, OP
Bussey, Frank R., 375 E. Main St., Bay Shore, N. Y., OP

Cady, Frederick J., Jr., 402 S. Jefferson Ave., Saginaw, Mich., OP
Cahill, John Edward, 196 Main St., Brockton 1, Mass., OP
Cahn, Herbert L., Medical Arts Bldg., Richland, Wash., ALR
Campbell, Allen Dryer, Jr. (Lt.), U.S.S. Haven AH-12, c/o Fleet Post Office, San Francisco, Calif., OP
Campbell, Edward Malcolm, 207 E. Watauga Ave., Johnson City, Tenn., OP
Campbell, George Maury, 1107 Hermann Professional Bldg., Houston 25, Texas, OP
Cannon, Edward Joseph (1st Lt.), Tokyo Army Hospital, 8059th AU, APO 1052, c/o Postmaster, San Francisco, Calif., OP
Caparosa, Ralph James (1st Lt.), 16th Field Hospital, APO 696, c/o Postmaster, New York, N. Y., ALR
Carriker, Frederick Ruthven (Capt.), Armed Forces Institute of Pathology, Washington 25, D. C., OP
Cavaliere, Joseph Thomas, 142 Joralemon St., Brooklyn 2, N. Y., OP
Cavins, Carl S., 804 W. Fifth St., Oxnard, Calif., ALR
Chamberlain, Douglas, 108 Interstate Bldg., Chattanooga, Tenn., ALR
Chandler, Clinton Beriah, McPherson Hospital, Durham, N. C., OP
Cinotti, Alfonse A., 3285 Hudson Blvd., Jersey City, N. J., OP
Clark, Charles Edward, 207 S. Commerce, Natchez, Miss., OP
Cleveland, Albert Francis, 615 Morgan Ave., Drexel Hill, Pa., OP
Cleveland, Crawford Hal, McWilliams Clinic, Gulfport, Miss., ALR
Cleveland, Willard Henry (Lt. Col.), U. S. Army Hospital, Fort Sill, Okla., OP
Clinckett, Frederick Robert, 233 Metcalfe St., Ottawa, Ont., Canada, OP
Clough, Joseph Messer, 101 Bay State Rd., Boston 15, Mass., OP
Cohen, Irwin Jerome, 54 Riverside Dr., New York 24, N. Y., OP
Cohen, M. Harry, 117 S. Second Ave., Mt. Vernon, N. Y., OP
Cohen, Morris Hyman, 331 Fulton St., Peoria, Ill., ALR
Coles, Robert S., 1148 Fifth Ave., New York 28, N. Y., OP
Colquhoun, Graham Fraser, 401 Security Bank Bldg., Battle Creek, Mich., OP
Colyear, Bayard Henry, Jr., 490 Post St., San Francisco 2, Calif., OP
Conley, James Patrick (Capt.), 141st General Hospital, APO 45, c/o Postmaster, San Francisco, Calif., ALR
Cook, Hugh Kenneth, 101 W. John St., Bay City, Mich., ALR
Cooley, Harold Niles, 620 Woodward Bldg., Birmingham, Ala., ALR
Cooper, Charles F., Jr., 490 Peachtree St. N. E., Atlanta 3, Ga., OP
Cooper, Robert Ray, 90 S. Ninth St., Minneapolis 2, Minn., OP
Copeland, Robert Lewis, Eye Clinic, U. S. Army Hospital, Fort George Meade, Md., OP
Cowper, Alexander Robertson, 543 Franklin St., Buffalo 2, N. Y., OP
Crain, Alfred Penn, Jr., 206 Physicians & Surgeons Bldg. East, Shreveport, La., OP
Crowley, Frederick Allison, 1008 N. Main St., Bloomington, Ill., OP

Cundy, Donald T., 1427 Medical Arts Bldg., Minneapolis 2, Minn., OP
Curtin, Brian Joseph, 1777 Grand Concourse, New York 53, N. Y., OP

Dardis, Walter Traynor, 230 Colorado Ave., Pueblo, Colo., OP
Davis, Jefferson, U. S. Naval Hospital, Portsmouth, Va., ALR
Dayton, Glenn Orville, Jr., 1052 W. Sixth St., Los Angeles 17, Calif., OP
Dean, Wynant, 300 Francis Bldg., Louisville 2, Ky., OP
de la Motte, G. Walter, 21 E. State St., Columbus 15, Ohio, OP
DeMere, McCarthy, 30 S. Bellevue, Memphis, Tenn., Pl S
Demshki, Andrew Edward, Jr., 322 Forward St., La Jolla, Calif., ALR
Dietrich, Herbert James, Jr., 1101 Beacon St., Brookline 46, Mass., ALR
Dobbs, Charles Eugene, 1304 Walker Ave., Houston 2, Texas, OP
Dolphin, Joseph Murray, 83 S. Franklin St., Wilkes Barre, Pa., OP
Doolittle, John Williams, 110 E. Main St., Madison 3, Wis., OP
Duszynski, Leonard Richard, 1073 Broadway, Buffalo 12, N. Y., OP
Dyson, Charles, 280 Queen's Ave., London, Ont., Canada, OP

Eareckson, Vincent O., Jr., 300 S. Hanson St., Easton, Md., OP
Ehrlich, Edward, 838 S. 13th St., Newark 8, N. J., ALR
Elizalde, Francisco J., Maipu 986, Buenos Aires, Argentina, OP
Ellison, Ellis, 617 Medical Arts Bldg., Minneapolis 2, Minn., ALR
Erler, Theodore George, Jr., 960 E. Green St., Pasadena 1, Calif., ALR

Failing, Willard N., Slocum-Dickson Clinic, 258 Genesee St., Utica 2, N. Y., OP
Farrington, Nolley Craft, 1407 S. Carrollton Ave., New Orleans 18, La., OP
Feamster, Felix C. (Lt. Col.), U. S. Army Hospital, Fort Benning, Ga., ALR
Fearon, Blair William, 308 Medical Arts Bldg., Toronto 5, Ont., Canada, ALR
Fee, George Alexander, 170 St. George St., Toronto, Ont., Canada, ALR
Feldman, Noah, 1060 Sanford Ave., Irvington 11, N. J., OP
Fenneman, Robert John, 609 Hulman Bldg., Evansville 18, Ind., OP
Fialkowski, Edward A., 761 Fillmore Ave., Buffalo 12, N. Y., OP
Fink, Philip Harold, 85-20 Lefferts Blvd., Kew Gardens 15, N. Y., OP
Fischer, Max J., 915 19th St. N. W., Washington 6, D. C., ALR
Fischer, Newton Duchan, N. C. Memorial Hospital, Chapel Hill, N. C., ALR
Flake, Carlyle Gregory, 300 Longwood Ave., Boston 15, Mass., ALR
Flynn, Gregory Edward, 144 Ponce de Leon Ave., Atlanta, Ga., OP
Fogo, Alden Eugene, 16 S. Henry St., Madison, Wis., ALR
Fokes, Robert Engram, Jr., 16 First Ave. S. W., Moultrie, Ga., OP
Fonda, Donald A., 114 Brookside Ave., Ridgewood, N. J., OP
Foote, Donovan Byrne, 422 N. Hastings Ave., Hastings, Neb., ALR
Forrest, Arnold William, 61 Maple Ave., White Plains, N. Y., OP
Fowler, Nathaniel Eugene, U.S.S. Baltimore (CA-68), c/o Fleet Post Office, New York, N. Y., OP
Fratkin, Max, 20 Plaza St., Brooklyn 17, N. Y., OP
Frayer, William C., 133 S. 36th St., Philadelphia 4, Pa., OP
Freedman, Robert Harrison, Partridge Road, Stamford, Conn., ALR
Freeman, Stanley Baruch, 147 E. 50th St., New York 22, N. Y., ALR
Friedman, Meyer H., 949 W. State St., Trenton 8, N. J., ALR
Frink, Berton F., Suite 2, M. & J. Nelson Bldg., McAllen, Texas, OP
Fuelling, James Louis (Comdr.), U. S. Naval Hospital, Bethesda, Md., OP
Fuhs, John Charles, 601 Union Trust Bldg., Cincinnati 2, Ohio, OP

Gaillard, Richard Allan (Comdr.), U. S. Naval Hospital, Bethesda, Md., ALR
Gans, Edward W., 280 MacArthur Blvd. West, Oakland 11, Calif., ALR
Garvin, William J., 80 Ashburton Ave., Yonkers, N. Y., OP
Gay, George Charles, 453 S. Soto St., Los Angeles 33, Calif., ALR
Gerlach, James J., 4 E. Eager St., Baltimore 2, Md., ALR
Gilliam, Robert Dale, U. S. Naval Hospital, Philadelphia, Pa., OP
Gillies, Ray O'Herin, Jr., Omaha Veterans Administration Hospital, Omaha, Neb., ALR
Goldberg, Harry F., 37 St. Andrews Pl., Yonkers 5, N. Y., OP
Goldware, Ross Russell, 436 N. Roxbury Dr., Beverly Hills, Calif., ALR
Gomillion, Jesse Durward, Merimax Bldg., Galveston, Texas, ALR

Good, Philip S., 112 Prospect St., Waterbury, Conn., OP
Good, Robert C., 112 Prospect St., Waterbury, Conn., OP
Goodall, Edwin Baker, 99 Bay State Rd., Boston 15, Mass., OP
Graham, John Herbert, 5500 Ridge Rd., Cleveland 29, Ohio, OP
Grana, Philip Charles, 401 Jenkins Bldg., Pittsburgh 22, Pa., OP
Gray, Harry William, 2017 Massachusetts Ave. N. W., Washington 6, D. C., OP
Green, Carl A., Box 655, Sampson Air Force Base Hospital, Geneva, N. Y., OP
Gregg, John Bailey, University Hospitals, Iowa City, Iowa, ALR
Guemmer, Gerald Edwin, 11 S. LaGrange Rd., LaGrange, Ill., ALR
Guest, Wilbur Cox, 309 Medical Arts Bldg., Winnipeg, Man., Canada, OP
Gurnee, Landon Harrison, 234 Madison St., Jefferson City, Mo., ALR

Haber, Norman, 518 Linwood Ave., Buffalo 9, N. Y., ALR
Haines, Robert William, 85 Jefferson St., Hartford 14, Conn., OP
Halden, Harry Edward, III, U.S. Public Health Service Hospital, Seattle 44, Wash., OP
Hales, Dean Wilson, 2955 Harrison Blvd., Ogden, Utah, ALR
Hall, Jack H. (Major), 3275 Medical Group, Parks Air Force Base, Calif., OP
Halle, Margaret Abrahamson, 4605 Minden Rd., Memphis 11, Tenn., OP
Hamm, William Gideon, Suite 206, 710 Peachtree St. N.E., Atlanta 3, Ga., Pl S
Hammett, Howard Hilt, Jr., Hammett Bldg., LaGrange, Ga., ALR
Hans, Clarence Louis, Jr., 807 Provident Bank Bldg., Cincinnati 2, Ohio, OP
Harcourt, Frederick Lee (Major), Walter Reed Army Hospital, Washington 12, D. C., ALR
Harding, M. Richard, 308 Hume Mansur Bldg., Indianapolis 4, Ind., OP
Harger, Robert William, 804 Hume Mansur Bldg., Indianapolis 4, Ind., OP
Hargett, Herbert P., 909 First Nat'l Bank Bldg., Springfield, Ohio, OP
Hargiss, James Leonard, 1059 Medical-Dental Bldg., Seattle 1, Wash., OP
Harris, Henry LeRew, 1136 W. Sixth St., Los Angeles 17, Calif., ALR
Harvey, Lyman Baxter, 13000 Maple Ave., Blue Island, Ill., OP
Hawkes, Alfred Kenneth, U.S. Naval Hospital, Memphis, Tenn., ALR
Heath, Scott Alexander, 664 Farmington Ave., Hartford 5, Conn., OP
Heinitsh, George, 125 E. Pennsylvania Ave., Southern Pines, N.C., ALR
Hermann, Ernest, 101 S. Main St., Canandaigua, N. Y., OP
Hermayer, Stephen, 124 S.E. First St., Evansville, Ind., OP
Herzon, Emanuel M., 164 Division St., Elgin, Ill., ALR
Heslin, Thomas Madden, 116 Livingston Ave., New Brunswick, N. J., OP
Hicks, Avery Morley, 490 Post St., Suite 1450, San Francisco 2, Calif., OP
Hill, John Mounce, 108 Bay State Rd., Boston 15, Mass., OP
Ho, Albert K. T., 1531 S. Beretania St., Honolulu 14, Hawaii, ALR
Hoffman, Richard A., 912 Union Central Bldg., Cincinnati 2, Ohio, OP
Hollingsworth, John Binford, 1904 Franklin St., Oakland 12, Calif., ALR
Holt, Thomas, Warrenton, N. C., ALR
Holtz, Marvin Jay, 211 Cherry Ave., Long Beach 2, Calif., OP
Homme, Owen H., 523 W. Sixth St., Los Angeles 14, Calif., ALR
Hooper, Robert James (Capt.), Box 142, U. S. Air Force Hospital, Keesler Air Force Base, Miss., OP
Hopkins, Julius H., 201 Medical Arts Bldg., Petersburg, Va., ALR
Hopkins, William G., 702 N. Main St., Pueblo, Colo., OP
Howard, Royal M., 100 Central Ave., Staten Island 1, N. Y., OP
Howe, Suzanne Adele Louise, 133 E. 58th St., New York 22, N. Y., ALR
Hugenberg, William Charles, 727 Nat'l Bank Bldg., Portsmouth, Ohio, ALR
Hughson, Donald Thomas, 208 E. Wisconsin Ave., Milwaukee 2, Wis., OP
Hulcher, Julius Charles, 1001 W. Franklin St., Richmond 20, Va., ALR
Hummel, Anton Joseph, 508 N. Grand Blvd., St. Louis 3, Mo., OP

Ioratti, John B., 30 Island Ave., Aurora, Ill., OP
Izen, Joseph Alfred, 704 Tatar, Pasadena, Texas, ALR

Jacobson, Jerry Hart (Capt.), 3320 Medical Group, Amarillo Air Force Base, Texas, OP
Jacobson, Philip, 462 William St., Williamsport, Pa., ALR
Jamieson, James Sidney, Korman Block, Kirkland Lake, Ont., Canada, ALR
Jayson, Arthur Victor, 2 Shelter Lane, Levittown, N. Y., ALR

Johnson, Frank Waters, 1435 Esplanade, Klamath Falls, Ore., OP
Johnson, George Edwin, Young Bldg., Dothan, Ala., ALR
Johnson, Jerry, 739-C, Maxwell Air Force Base, Ala., OP
Johnston, Cecil Frederick, Bluefield Sanitarium, Bluefield, W. Va., ALR
Johnston, Kenneth Clayton, 700 N. Michigan Ave., Chicago 11, Ill., ALR
Johnston, William Harry, 505 Pleasant St., St. Joseph, Mich., OP
Jones, Millard Filmore, 2064 Park St., Jacksonville 4, Fla., ALR
Jones, Rheim Magleby, 1050 Memorial Dr., Idaho Falls, Idaho, OP
Jones, Wendell Leroy, 216 Dixon Bldg., Missoula, Mont., ALR

Kane, David Saul, 109 N. Wabash Ave., Chicago 2, Ill., OP
Kantar, Bruce Leo, 2925 Dean Blvd., Minneapolis, Minn., OP
Kapustiak, Wendell A., 3451 W. 63rd St., Chicago 29, Ill., OP
Karakashian, Nubar Arakel, 539 E. Allegheny Ave., Philadelphia 34, Pa., OP
Kaufmann, Mark I. H., 1540 Crescent St., Montreal 25, Que., Canada, OP
Kearns, Thomas P., Mayo Clinic, Rochester, Minn., OP
Keipp, James Vernon, 110 Pine Ave., Long Beach 2, Calif., OP
Keith, Frank R., Suite 514, Adams Bldg., Port Arthur, Texas, OP
Kelly, Robert Gordon, 113 St. Clair Ave. West, Toronto, Ont., Canada, OP
Kenet, David S., 417 Marble Arcade, Lakeland, Fla., ALR
Kenney, LeRoy Linus (Comdr.), U. S. Naval Hospital, Pensacola, Fla., ALR
Keppen, Ford F., 314 Tenth St., Richmond 2, Calif., OP
Kestenbaum, Alfred, 470 Park Ave., New York 22, N. Y., OP
Killian, E. Camille Hill, 211 S. Third St., Logansport, Ind., OP
Killian, Edgar William, 211 S. Third St., Logansport, Ind., ALR
King, Joseph Wheeler, 267 Citizens Bank Bldg., Anderson, Ind., ALR
Kirchgeorg, Clemens, 409 E. Wisconsin Ave., Neenah, Wis., OP
Kirschman, Robert Edward, 215 Frisco Bldg., Joplin, Mo., ALR
Kissling, Arthur C., Jr., 1324 W. Wisconsin Ave., Milwaukee 3, Wis., OP
Kitchen, Calvin Blaine, 150 E. Broad St., Columbus 15, Ohio, OP
Kleiner, David, 1 Chain Lane, Hicksville, N. Y., ALR
Knauer, William Jerome, Jr., 206 W. Forsyth St., Jacksonville 1, Fla., OP
Kobley, Donald E., U. S. Army Hospital, Fort Hood, Texas, OP
Koller, Robert Louis, 4959 Excelsior Blvd., Minneapolis 16, Minn., ALR
Kratka, William Herbert, Medical Arts Bldg., Wilmington 2, Del., OP
Kratz, Richard P., 6356½ Van Nuys Blvd., Van Nuys, Calif., OP
Kring, Harvey Wendell, 305 E. Douglas St., Roseburg, Ore., ALR
Krucoff, Morris E., 917 20th St. N. W., Washington 6, D. C., ALR
Kushi, Harold S., Kahului, Maui, Hawaii, OP

Lamp, Clyde Benjamin, Jr., 8102 Jenkins Arcade, Pittsburgh 22, Pa., ALR
Landon, H. Robert, 148-46 87th Ave., Jamaica 2, N. Y., ALR
Landowski, Jules, 123 E. 83rd St., New York 28, N. Y., OP
Landrigan, Frederick Laurence, 520 Commonwealth Ave., Boston 15, Mass., OP
Larkin, Ernest Waddill, Jr., 123 W. Third St., Greenville, N. C., OP
Lathrop, George E. (Lt. Col.), Tokyo Army Hospital, 8059 AU, APO 1052, c/o Postmaster, San Francisco, Calif., OP
Lautz, Herbert Arthur, 112 Rimbach St., Hammond, Ind., ALR
Layton, George A., 114 E. 54th St., New York 22, N. Y., OP
Lehman, Roger Hays, Veterans Administration Center, Wood, Wis., ALR
LeMaster, Theodore Ralph, 23 E. Ohio St., Room 805, Indianapolis 4, Ind., OP
Leonard, Thomas P., 132 S. Water St., Decatur, Ill., ALR
Levine, Julius, 1030 McKeever Ct., Hayward, Calif., OP
Lewallen, William M., Jr., Veterans Administration Hospital, McKinney, Texas, ALR
Lewis, Miles L., Jr., Ochsner Clinic, New Orleans 15, La., ALR
Lieberman, Howard Leslie, 2800 Milwaukee Ave., Chicago 18, Ill., OP
Lillie, John Canfield, Mayo Clinic, Rochester, Minn., ALR
Lindenberg, Paul, 115 E. 61st St., New York 21, N. Y., ALR
Lineback, Merrill Ingham, 113 14th St. N. E., Atlanta, Ga., ALR
Lingeman, Raleigh Eugene, 411 Hume Mansur Bldg., Indianapolis 4, Ind., ALR
Linger, Harry Teter, 610 Goff Bldg., Clarksburg, W. Va., OP

Little, Lewis Neville, 233 Metcalfe St., Ottawa, Ont., Canada, ALR
Liva, Vinicio G., 5 Pangborn Pl., Hackensack, N. J., OP
Lockhart, Henry Bent, 415 Westminster Trust Bldg., 713 Columbia St., New Westminster, B. C., Canada, ALR
London, William, 3020 29th St., Long Island City 2, N. Y., OP
Lownik, Felix Joseph, State Bank Bldg., Freeport, Ill., ALR
Lyda, Wood, 812 Maison Blanche Bldg., New Orleans 16, La., OP

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Madsen, George J., 1412 Fremont, Las Vegas, Nev., OP
Mandeville, Jack W., 1212 S. 11th St., Tacoma 3, Wash., OP
Marcus, Arthur Alvin, 30 E. 40th St., New York 16, N. Y., OP
Markham, Mary H., 135 E. 64th St., New York 21, N. Y., ALR
Marshall, Victor M., 1930 Wilshire Blvd., Los Angeles 57, Calif., OP
Martinucci, August, 58 N. Chicago St., Joliet, Ill., ALR
Masaryk, Joseph A., 100 Fourth St., Barbeton, Ohio, OP
McCann, Donald Somers, 7 N. Main St., Attleboro, Mass., OP
McClure, G. David, 565 Eastern Parkway, Louisville 8, Ky., OP
McCrory, Thomas Milton, 206 Medical Arts Bldg., Dallas 1, Texas, OP
McCusker, Wilfrid D., 201 Church St., Newark, N. Y., OP
McDonald, James E., 120 N. Oak Park Ave., Oak Park, Ill., OP
McEneaney, Joseph Patrick, 301 Essex St., Lawrence, Mass., ALR
McFarland, Kenneth T. H., 5095 Jenkins Arcade, Pittsburgh 22, Pa., OP
McFarland, Paul E., Park Central Medical Bldg., 550 W. Thomas Rd., Phoenix, Ariz., OP
McGee, Harry B., 1705½ Third St., Bay City, Mich., OP
McGinnis, Lyle B., 1113 Sixth Ave., Huntington, W. Va., ALR
McGrath, William Powers, 250 Pelissier St., Windsor, Ont., Canada, OP
McIver, Harold Thomas, 444 E. Main St., Jackson, Tenn., ALR
McIver, John Mark, 59 Coddington St., Quincy 69, Mass., OP
McMath, William Bates, 142 S. Jackson St., Americus, Ga., OP
McNelis, Francis Leo, 311 Angell St., Providence 6, R. I., ALR
McWilliams, John Robert, 1567 Westview Dr. N. E., Warren, Ohio, OP
Meisenback, Albert Edward, Jr., 300 Medical Arts Bldg., Dallas 1, Texas, OP
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Merrill, Horace Ross, 3251 Fourth Ave., San Diego 3, Calif., OP
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Minatoya, Wilfred Takeo, 1003 Pensacola St., Honolulu 14, Hawaii, OP
Moon, Cary Nelson, Jr., 104 E. Market St., Charlottesville, Va., ALR
Moore, Loran Paschal, 405 St. Ann St., Owensboro, Ky., OP
Morris, Jean Willard, 247 Johnson Bldg., Muncie, Ind., OP
Morrow, Rufus Clegg, 291 College St., Burlington, Vt., ALR
Morse, Harry Russell, 144 Ponce de Leon Ave. N. E., Atlanta, Ga., ALR
Moses, Jacob, 150 E. Broad St., Columbus 15, Ohio, OP
Moses, Robert Abram, 110½ S. Third St., Delavan, Wis., OP
Moskowitz, Herbert Leon, Veterans Administration Hospital, East Orange, N. J., OP
Moss, Ennis Edward, Jr., 2845 Stratford Rd., Columbia, S. C., OP
Mould, Ward L., 131 Fulton Ave., Hempstead, L. I., N. Y., OP
Mueller, George Christopher, 630 Park Ave., New York 21, N. Y., ALR
Murphy, Edward Underhill, 908 Hulman Bldg., Evansville, Ind., OP
Murphy, Robert Harold, 4753 N. Broadway, Chicago 40, Ill., OP
Murrah, William Fitzhugh, Jr., 1720 Exchange Bldg., Memphis 3, Tenn., OP

Nagle, Frank O., Jr., 37 S. 20th St., Philadelphia 3, Pa., OP
Naramore, Malta Lane, 625 Woodward Bldg., Birmingham 3, Ala., ALR
Newell, Edward Alphonso, 1511 N. Beckley, Dallas 8, Texas, ALR

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Norman, Wayman Bowers, Hurst Clinic, Longview, Texas, ALR

O'Connor, Edward Francis, 1 Chicago Ave., Oak Park, Ill., OP
Oliver, Dalton Senter, 419 Franklin St., Clarksville, Tenn., OP
Olson, Burton G., McCannel Clinic, Minot, N. D., OP
Olson, Nelse Olof, U. S. Naval Hospital, Bremerton, Wash., OP
Osborn, Robert Wallace, 9½ S. Main St. (Box 1666), McAllen, Texas, ALR

Pang, Herbert George, 52 S. Vineyard St., Honolulu 13, Hawaii, OP
Paprocki, Theodore Felix, 405 Caroline St., Ogdensburg, N. Y., OP
Parker, Willard, 2020 E. 93rd St., Cleveland 6, Ohio, ALR
Patterson, Harold Calvin, 8 West St., Danbury, Conn., OP
Patz, Arnall, 920 St. Paul St., Baltimore 2, Md., OP
Paulson, Hubert Oscar, 508 Sharp Bldg., Lincoln 8, Neb., OP
Payton, Calvin Worth, Hurst Clinic, Longview, Texas, OP
Pearce, Rowan C., Jr., 514 Cooper St., Camden 2, N. J., ALR
Pepe, Salvatore A., 1418 W. State St., Trenton, N. J., ALR
Perkins, Henry Roscoe, 810 Southern Finance Bldg., Augusta, Ga., OP
Perley, Edward Peter, 524 E. 20th St., New York 9, N. Y., OP
Persing, Harry M., Jr., 2314 Broad Ave., Altoona, Pa., OP
Pfister, Raymond L., 2600 Union Central Bldg., Cincinnati 2, Ohio, ALR
Phillipson, Chester, 7411 Third Ave., Detroit 2, Mich., ALR
Porter, Harry P., 409 Medical Arts Bldg., Cathedral and Read Sts., Baltimore 1, Md., ALR
Potts, Albert M., 2065 Adelbert Rd., Cleveland 6, Ohio, OP
Pou, Jack Wendell, 1513 Line Ave., Shreveport, La., ALR
Poweleit, Alvin Charles, 637 Scott St., Covington, Ky., OALR
Power, John Leahy, 2001 Fourth Ave., San Diego 1, Calif., OP
Powers, William Hugh, 1011 Lake St., Oak Park, Ill., ALR
Pratt, Fred John, Ninth Medical Detachment, APO 108, c/o Postmaster, New York, N. Y., OP
Privett, Lowell Bernard, 509 First Nat'l Bank Bldg., Boise, Idaho, OP
Prough, Wendell Arthur, 326 N. Laurel Ave., Ontario, Calif., OP

Rachlin, Maxwell, 1695 S. Brentwood, Brentwood 17, Mo., OP
Radoff, Marvin L., 2 Lakeside Dr. West, Levittown, Pa., ALR
Rawdon, Robert E., 33 W. Third St., Lewistown, Pa., ALR
Reed, George F., 243 Charles St., Boston 14, Mass., ALR
Regan, John Edward, 82 Broadway, Taunton, Mass., OP
Reich, August, 450 Sutter St., San Francisco 8, Calif., ALR
Rider, Mitchell Burns, 1545 Addison Ave. East, Twin Falls, Idaho, OP
Riffenburgh, Ralph Sidney, 640 S. Kingshighway, St. Louis 10, Mo., OP
Rigg, Marilyn Martin, 512 E. Penn St., Philadelphia 44, Pa., OP
Ringenberg, Jordan Chris, 420 Medical Arts Bldg., Grand Rapids 2, Mich., ALR
Ripple, Paul H., 616 N. Lime St., Lancaster, Pa., OP
Roberts, James Eugene, 727 W. Seventh St., Los Angeles 17, Calif., OP
Robertson, William Craig, 1039 Forrest Ave., Gadsden, Ala., OP
Robison, James Travis, Jr., 411 Nichols Rd., Kansas City 13, Mo., OP
Rockowitz, Erwin Harry (1st Lt.), Rodriguez Army Hospital, San Juan (Fort Brooke), Puerto Rico, ALR
Root, Amos B., Jr., 233 A St., San Diego 1, Calif., OP
Rubin, Maurice Harvey, 2164-1 U. S. Army Hospital, Fort Eustis, Va., OP
Rubin, Wallace, 305 Baronne St., New Orleans 12, La., ALR
Rundles, Walter Zell, Jr., 304 National Bldg., Flint 3, Mich., OP
Rupp, Robert Allen, 822 Fayette Nat'l Bank Bldg., Uniontown, Pa., ALR
Rutberg, Franklin Leon, 1658 E. Washington Lane, Philadelphia 38, Pa., ALR
Ryan, Emmett J. (Capt.), U. S. Army Hospital, Camp Stoneman, Calif., OP
Ryan, Ralph W., National Institutes of Health, Bethesda 14, Md., OP
Ryan, Stephen Joseph, U. S. Naval Hospital, Bethesda, Md., OP

Samuels, S. Lawrence, 1111 Park Ave., Plainfield, N. J., OP
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Schachne, Lewis, U. S. Naval Hospital, St. Albans, N. Y., OP

Schardt, Walter Mallory, 935 Main St., Manchester, Conn., OP
Schertz, Truman B., 902 Edmond St., St. Joseph, Mo., OP
Schoel, Robert Edward, 211 S. Saratoga St., New Orleans 12, La., OP
Schrimpf, William John, U. S. Air Force Hospital, Lackland Air Force Base, San Antonio, Texas, ALR
Schultz, Arthur Edward, 119 E. Grand River Ave., East Lansing, Mich., OP
Schwartz, Frank Edwin, U. S. Army Hospital, Camp Atterbury, Ind., OP
Scott, David Henry, 1 Monument Sq., Beverly, Mass., OP
Seely, Richard H., 104 N. Clinton Ave., Trenton 9, N. J., OP
Settembrini, Charles Louis, 689 Gramatan Ave., Mt. Vernon, N. Y., OP
Shaw, Harry Edward, 8024 Jenkins Arcade, Pittsburgh 22, Pa., OP
Shaw, Jules Harold, 510 Commonwealth Ave., Boston 15, Mass., OP
Shepley, Felix Rhodes, The Medical and Surgical Clinic, Hamilton Bldg., Wichita Falls, Texas, ALR
Shulman, Albert George, 91 Oak St., Binghamton, N. Y., OP
Shuster, Donald Williams (1st Lt.), U. S. Air Force School of Aviation Medicine, Box 169, Randolph Air Force Base, Texas, ALR
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Sibbitt, Joseph W., 300 E. Kirkwood, Bloomington, Ind., ALR
Siemon, Glenn, 2014 17th St., Bakersfield, Calif., OP
Silver, M. David, 200 Central Park South, New York 19, N. Y., OP
Sirota, Harvey H., 1128 Main St., East Hartford, Conn., ALR
Slabaugh, Carlyle B., 1733 Brownlee St., Corpus Christi, Texas, OP
Smart, Gardner Ford, 801 City Bldg., Asheville, N. C., ALR
Smith, Arthur Cook, Jr., 410 W. Church St., Elmira, N. Y., OP
Snip, Russell Thorn, 505 Howard St., San Antonio 1, Texas, OP
Snow, Virgil Crosby, 2942 Fresno St., Fresno, Calif., OP
Snyderman, Sanford Charles, 629 Medical Center Bldg., Fort Wayne, Ind., ALR
Solomon, Alex E., 8057 Beverly Blvd., Los Angeles 48, Calif., ALR
Spector, Martin, 2148 E. Cumberland St., Philadelphia 25, Pa., ALR
Sperling, Harry H., 1245 Glendon Ave., Westwood Village, Los Angeles 24, Calif., ALR
Steele, Burns Charles, 265 E. Orange Grove Ave., Burbank, Calif., ALR
Steiman, Solomon Eliah, 23 Broad St., Lynn, Mass., OP
Stephens, Vernon Dean, 9 Buttles Ave., Columbus 8, Ohio, OP
Stevens, Sydney Louis, 23 E. Ohio St., Suite 303, Indianapolis 4, Ind., ALR
Stoolfire, Arthur W., The Medical Arts Clinic, Merchants & Planters Bank Bldg., Sherman, Texas, ALR
Strueh, Paul Edward, 124 S. E. First St., Evansville, Ind., ALR
Stuart, Robert Leo, Foley Bldg., La Grande, Ore., OP
Stuart, Royal Eppes (Lt.), U. S. Naval Hospital, Newport, R. I., ALR
Suitor, Jesse Hale, U. S. Naval Hospital, Memphis, Tenn., OP
Sumner, Robert Holloway, 571 W. Seventh St., San Pedro, Calif., ALR
Swett, Wilber Frank, 490 Post St., San Francisco 2, Calif., OP
Sykowski, Peter Stephen, 1330 Union St., Schenectady 8, N. Y., OP
Szakalun, Leo Stanley (Major), U. S. Army Hospital, Fort Ord, Calif., OP
Szewczyk, Thaddeus S., Metropolitan Bldg., East St. Louis, Ill., OP

Taylor, Thomas J., 319 Security Bldg., Olympia, Wash., ALR
Tebbet, Royce Dwain, 218 Wyoming Nat'l Bank Bldg., Casper, Wyo., OP
Terrill, Richard Wingate, 455 Lincoln Bank Tower, Fort Wayne 6, Ind., OP
Thee, Earl John, Jr., Wadsworth GM&SH, Wilshire and Sawtelle Blvds., Los Angeles 25, Calif., ALR
Thomas, Edwin Bentley, U. S. Public Health Service Hospital, Staten Island 4, N. Y., OP
Thompson, Errol Aldwyn, 154 W. 119th St., New York 26, N. Y., ALR
Tomarelli, Raymond C., 807 Empire Bldg., Pittsburgh 22, Pa., OP
Tresley, Jack, 6 N. Hamlin Ave., Chicago 24, Ill., OP
Trichter, Benjamin, 645 Ocean Ave., Brooklyn 26, N. Y., OP
Trolan, Howard, 519 Soquel Ave., Santa Cruz, Calif., OP
Turgeon, Leo Victor, Jr., 3741 Stocker St., Los Angeles 8, Calif., ALR
Turnblacer, Charles B., 340 N. Main St., Butler, Pa., ALR

Vaughan, Daniel George, Jr., 220 Meridian Rd., San Jose, Calif., OP
Venable, Howard Phillip, 11 N. Jefferson Ave., St. Louis 3, Mo., OP

Vertuno, John W., 231 W. Washington St., Chicago 6, Ill., OP
Vickery, Robert David, 237 Aquila Ct., Omaha 2, Neb., OP
Volk, Myron, 18599 Lakeshore Blvd., Euclid, Ohio, OP

Waldron, Edward Louis, 312 Bellevue Ave., Trenton, N. J., ALR
Wallace, Thomas Hendrix, 1205 Cleveland St., Clearwater, Fla., ALR
Wallber, Fred E., 350 A St., Idaho Falls, Idaho, OP
Wallenborn, Peter A., Jr., 209 Medical Arts Bldg., Roanoke, Va., ALR
Walsh, John K., 609 Main St., Orange, Texas, OP
Ward, Clarence Vincent, Lehmann Bldg., Peoria, Ill., OP
Wattleworth, Kent Lincoln, 106 N. Silver St., Olney, Ill., ALR
Watts, James Harvey, Jr., U. S. Naval Hospital, Mare Island, Vallejo, Calif., ALR
Weidemann, John Albert, 6545 W. North Ave., Oak Park, Ill., ALR
Weille, Francis Lee, 247 Commonwealth Ave., Boston 8, Mass., ALR
Welt, Milton, 295 Clinton Ave., Brooklyn 5, N. Y., OP
Wetzig, Paul Carl, 830 N. Tejon St., Colorado Springs, Colo., OP
Whaley, James Black, 170 St. George St., Toronto, Ont., Canada, ALR
Whistler, Carl William, Veterans Administration Hospital, Denver, Colo., ALR
White, Frederick Dashiell, Bluefield Sanitarium, Bluefield, W. Va., OP
Whittington, James C., Exchange Bldg., Eastland, Texas, ALR
Wickerham, Earl Phillips, Jr., 807 Peoples East End Bldg., Pittsburgh 6, Pa., OP
Wiener, Howard E., 285 Central Ave., Lawrence, N. Y., OP
Wilf, Harold, 1647 N. 29th St., Philadelphia 21, Pa., ALR
Williams, Frederick Davis, 1 Atlantic St., Stamford, Conn., OP
Williams, Mortimer Lee, U. S. Army Hospital, Fort Ord, Calif., ALR
Wilson, Charles F., Pikeville, Ky., OP
Winter, Frank Counsel, Memorial Hospital, Chapel Hill, N. C., OP
Wippermann, Edgar David, 6407th Hospital Group, APO 323-2, c/o Postmaster, San Francisco, Calif., ALR
Wissner, Ulrich Carl, 1401 S. Hope St., Los Angeles 15, Calif., OP
Wolf, Earl George, 1825 N. Palafox St., Pensacola, Fla., ALR
Wones, Edward M., Jr., 215 N. Sheridan Rd., Waukegan, Ill., OP
Works, Royal Leone, 947 W. Eighth St., Los Angeles 17, Calif., OALR
Wortham, Edwin, 215 Estudillo Ave., San Leandro, Calif., OP

Yates, C. Dwight, 1213 14th St., Modesto, Calif., ALR
Young, Robert Lawrence, 708 Broadway, Gary, Ind., OP

Zoller, Harry, 1109 Pere Marquette Bldg., New Orleans 12, La., ALR
Zuidema, Jacob J., 1002 Ninth St., Greeley, Colo., ALR
Zwerling, Martin Herbert, 375 E. Main St., Bay Shore, L. I., N. Y., ALR

The following candidates have qualified for Fellowship in the Academy except in some single detail—certification, membership in a local medical society, or the signatures of sponsors. These deficiencies are expected to be made up before the Annual Business Meeting.

Droege, Fred D., 208 Vanskike Bldg., Sarasota, Fla.
Harper, John Y., 849 Fifth St., Port Arthur, Texas
Horsley, George E., Madison Sanitarium and Hospital, Madison College, Tenn., ALR
Little, C. Robertson, 406 Medical Professional Bldg., Corpus Christi, Texas
Sherman, Alfred G., Hyde Bldg., Albert Lea, Minn., OP
Thomas, Richard Louis, Suite 601-605, Stuart Bldg., Lincoln 8, Neb.
Urrets-Zavalia, Alberto, Jr., 27 de Abril 255, Cordoba, Argentina, S. A., OP

PROGRAM IN BRIEF

1953 Meeting — Chicago

SATURDAY, OCTOBER 10, 1953

9:00 a.m.—Council Meeting—Private Dining Room 4

8:00 p.m.—Special Scientific Program: Otosclerosis Study Group—Private Dining Room 14

SUNDAY, OCTOBER 11, 1953

9:00 a.m.—Registration—Foyer to Grand Ball Room

9:00 a.m.—Ladies' Registration—Private Dining Rooms 15 and 16

9:00 a.m.—Official Opening of Exhibits:
Scientific—Red Lacquer Room
Technical—Exhibition Hall and Red Lacquer Room

10:00 a.m.—Teachers' Section: Ophthalmology—Crystal Room

10:00 a.m.—Teachers' Section: Otolaryngology—Private Dining Room 14

12:30 p.m.—Home Study Courses Faculty Luncheon—Private Dining Room 9

4:30 p.m.—President's Reception—Grand Ball Room

8:00 p.m.—Special Scientific Program: Committee on Conservation of Hearing and Otosclerosis Study Group—Grand Ball Room

8:00 p.m.—Special Scientific Program: Joint Committee on Industrial Ophthalmology—Private Dining Room 14

MONDAY, OCTOBER 12, 1953

9:00 a.m.—Orthoptic Technicians Instruction Courses—Seventh Floor and Private Dining Room 17

9:30 a.m.—Joint Scientific Session—Grand Ball Room

10:15 a.m.—Orthoptic Technicians Instruction Courses—Seventh Floor and Private Dining Room 17

11:30 a.m.—Orthoptic Technicians Instruction Courses—Seventh Floor and Private Dining Room 17

2:00 p.m.—Section on Otolaryngology: Scientific Papers and Motion Pictures—Grand Ball Room

2:00 p.m.—EYE Instruction Courses—Seventh Floor and Private Dining Rooms 14 and 17

3:15 p.m.—EYE Instruction Courses—Seventh Floor and Private Dining Rooms 14 and 17

4:30 p.m.—EYE Instruction Courses—Seventh Floor and Private Dining Room 17

6:30 p.m.—Alumni Dinners

9:00 p.m.—Evening of Diversion

TUESDAY, OCTOBER 13, 1953

9:00 a.m.—Section on Ophthalmology: Scientific Papers and Motion Pictures—Grand Ball Room

9:00 a.m.—ENT Instruction Courses—Seventh Floor

9:00 a.m.—Orthoptic Technicians Instruction Courses—Seventh Floor and Private Dining Room 17

10:15 a.m.—ENT Instruction Courses—Seventh Floor

10:15 a.m.—Orthoptic Technicians Instruction Courses—Seventh Floor and Private Dining Room 17

11:30 a.m.—ENT Instruction Courses—Seventh Floor

11:30 a.m.—Orthoptic Technicians Instruction Courses—Seventh Floor and Private Dining Room 17

12:30 p.m.—Technical Exhibitors Luncheon—Private Dining Room 9

2:00 p.m.—Section on Otolaryngology: Scientific Papers and Motion Pictures—Grand Ball Room

2:00 p.m.—EYE Instruction Courses—Seventh Floor and Private Dining Room 17

3:15 p.m.—EYE Instruction Courses—Seventh Floor and Private Dining Room 17

4:30 p.m.—EYE Instruction Courses—Seventh Floor and Private Dining Room 17

5:30 p.m.—Dinner Meeting of International Association of Eye, Ear, Nose and Throat Society Secretaries—Private Dining Room 18

8:00 p.m.—Special Scientific Program: American Orthoptic Council and American Association of Orthoptic Technicians—Private Dining Room 14

8:00 p.m.—Special Scientific Program: Committee on Plastic Surgery—Grand Ball Room

WEDNESDAY, OCTOBER 14, 1953

9:00 a.m.—Section on Ophthalmology: Scientific Papers and Motion Pictures—Grand Ball Room

9:00 a.m.—ENT Instruction Courses—Seventh Floor

10:15 a.m.—ENT Instruction Courses—Seventh Floor

11:30 a.m.—ENT Instruction Courses—Seventh Floor

12:45 p.m.—Council Meeting Luncheon—Private Dining Room 5

2:00 p.m.—Section on Otolaryngology: Scientific Papers and Motion Pictures—Grand Ball Room

2:00 p.m.—EYE Instruction Courses—Seventh Floor and Private Dining Room 17

3:15 p.m.—EYE Instruction Courses—Seventh Floor and Private Dining Room 17

4:30 p.m.—EYE Instruction Courses—Seventh Floor and Private Dining Room 17

7:00 p.m.—Annual Dinner—Grand Ball Room

THURSDAY, OCTOBER 15, 1953

9:00 a.m.—Section on Ophthalmology: Scientific Papers and Motion Pictures—Grand Ball Room

9:00 a.m.—ENT Instruction Courses—Seventh Floor

10:15 a.m.—ENT Instruction Courses—Seventh Floor

11:30 a.m.—ENT Instruction Courses—Seventh Floor

12:45 p.m.—Faculty Luncheon—Private Dining Room 14

2:00 p.m.—Section on Otolaryngology: Scientific Papers and Motion Pictures—Grand Ball Room

2:00 p.m.—EYE Instruction Courses—Seventh Floor

3:15 p.m.—EYE Instruction Courses—Seventh Floor

4:30 p.m.—EYE Instruction Courses—Seventh Floor

5:30 p.m.—Business Meeting—Crystal Room

8:00 p.m.—Motion Pictures — Grand Ball Room

FRIDAY, OCTOBER 16, 1953

9:00 a.m.—Section on Ophthalmology: Scientific Papers and Motion Pictures—Grand Ball Room

9:00 a.m.—Joint Scientific Session with American Society of Ophthalmologic and Otolaryngologic Allergy—Private Dining Room 14

2:00 p.m.—Joint Scientific Session with American Society of Ophthalmologic and Otolaryngologic Allergy—Grand Ball Room

SCIENTIFIC SECTIONS

JOINT SESSION

Monday, October 12, 1953

9:30 a.m.

GRAND BALL ROOM

Presiding Officer:
FREDERICK C. CORDES, M.D.
President

Secretary:
KENNETH L. ROPER, M.D.

Call to Order.....The President

Announcements.....The Secretary

Introduction of the Guest of Honor, Lawrence T. Post, M.D., and Other Distinguished Guests.....The President

Address by the President

The Certificate of the American Board of Ophthalmology: An Evaluation

Address by the Guest of Honor

The Art of the Practice of Medicine as It Applies to Ophthalmology

Address by the Guest Speaker

The Changing Face of Medicine

Mr. Frank W. Law
London, England

Presentation of Honor Keys.....The President

James B. Costen, M.D., St. Louis Mo.
A. Gerard DeVoe, M.D., New York, N. Y.
J. Conrad Gemeroy, M.D., Detroit, Mich.
John F. Gipner, M.D., Rochester, N. Y.
Harry N. Glick, M.D., St. Louis, Mo.
Arthur L. Juers, M.D., Louisville, Ky.
Varaztad H. Kazanjian, M.D., Boston, Mass.
W. Howard Morrison, M.D., Omaha, Neb.
Frank W. Newell, M.D., Chicago, Ill.
Donald H. O'Rourke, M.D., Denver, Colo.
Harold G. Scheie, M.D., Philadelphia, Pa.

SYMPOSIUM: MALUNITED FRACTURES OF THE ZYGOMA

Presented by the Committee on Plastic Surgery
John M. Converse, M.D.
Wendell L. Hughes, M.D.
Co-Chairmen

Damage to Bones and Soft Tissues

Varaztad H. Kazanjian, M.D.
Boston, Mass.

Bony Depressions, Particularly Depressions of the Floor Of the Orbit

Alston Callahan, M.D.
Birmingham, Ala.

The Correction of Diplopia After Fractures of the Orbit

Edmund B. Spaeth, M.D.
Philadelphia, Pa.

Repair of the Deformity

Reed O. Dingman, M.D.
Ann Arbor, Mich.

BY INVITATION

AUTHORS OF PAPERS

All slides or films must be in the hands of the projection operator before the opening of each session. Please be prepared to submit papers to the recorder immediately following presentation.

SECTION ON OTOLARYNGOLOGY

Monday, October 12, 1953
2:00 p.m.
GRAND BALL ROOM

Presiding Officer:
THOMAS C. GALLOWAY, M.D.
Vice-President

Secretary:
HOWARD P. HOUSE, M.D.

PRESENTATION OF NEW INSTRUMENTS AND NEW TECHNICS

2:10 p.m.

EFFECT OF PREGNANCY ON OTOSCLEROSIS

(Sponsored by the Otosclerosis Study Group)

Theodore E. Walsh, M.D.
St. Louis, Mo.

Discussers:
George E. Shambaugh, Jr., M.D.
Chicago, Ill.
Moses H. Lurie, M.D.
Boston, Mass.

Although it has been generally supposed that pregnancy is responsible for a further loss of hearing in otosclerosis it has been the experience of this author that a positive relationship could be established by history in only a small percentage of cases.

As a considerable number of women who have undergone fenestration have later become pregnant, a review of the subsequent hearing in both the fenestrated and unfenestrated ears is presented in order to evaluate, if possible, the effect of pregnancy on otosclerosis.

2:50 p.m.

THE ANATOMY AND PATHOLOGY OF THE EAR DRUM
(A Motion Picture)

Paul H. Holinger, M.D.
and
Kenneth C. Johnston, M.D.
BY INVITATION
Richard A. Buckingham, M. D.
BY INVITATION
Chicago, Ill.

The anatomy and pathology of the ear drum are shown as seen through the ear speculum and the otoscope. The normal drum membrane is first demonstrated and then various inflammatory processes, perforations and other drum pictures are shown. In the absence of the drum as in a large perforation, the contents of the middle ear are visualized.

3:15 p.m.

VOCAL CORD NEUROSIS

Paul J. Moses, M.D.
San Francisco, Calif.
BY INVITATION

Discusser:
Paul Moore, M.D.
Evanston, Ill.
BY INVITATION

The laryngologists see in their practice a large percentage of functional disturbances of the larynx which are not visible to the eye, but the vocal changes of which are audible to the ear. This paper proposes to outline how vocal dynamics express psychodynamics. Different forms of neurosis have their specific and characteristic vocal expression. A new approach to vocal analysis is demonstrated, helping to explain these specific correlations between voice and neurosis; for instance, inspiratory voice, persistent falsetto voice, aphonia, and so-called myasthenia of the professional speaker. This paper will prove that neither voice rest nor the routine voice exercises help unless the true cause for these dysfunctions is discovered. Then the therapy needed reveals itself. On the other hand, all these disturbances cause changes of laryngeal function which usually reveal themselves in local irritation, too often confused with inflammatory processes. Proper evaluation is possible only if the psychosomatic mechanism can be analyzed. This often saves the patient from unnecessary referrals to voice teachers or psychiatrists, as well as from wrong medication.

3:55 p.m.

SYMPOSIUM: POSTNASAL DISCHARGE

Arthur W. Proetz, M.D.
St. Louis, Mo.

Lawrence R. Boies, M.D.
Minneapolis, Minn.

Lewis F. Morrison, M.D.
San Francisco, Calif.

While postnasal discharges may be simply classified as serous, mucous, purulent or hemorrhagic, the underlying factors which cause them number a score or more. The obvious ones will be superficially dealt with; those less apparent and hence more difficult of diagnosis will be discussed by all three participants with special emphasis on management.

SECTION ON OPHTHALMOLOGY

Tuesday, October 13, 1953
9:00 a.m.
GRAND BALL ROOM

Presiding Officer:
HARVEY E. THORPE, M.D.
Vice-President

Secretary:
KENNETH L. ROPER, M.D.

ARACHNODACTYLY (MARFAN'S SYNDROME) WITH ECTOPIA LENTIS AND MYOPIA
(A Clinicopathologic Case Report)

Merrill J. Reeh, M.D.
and
William L. Lehman, M.D.
BY INVITATION
Portland, Ore.

A typical case of arachnodactyly in a 25-year-old white male who died suddenly of a dissecting aneurysm is presented. He had ectopia lentis and myopia with excellent vision afforded by corrective lenses. Histologic study of the eyes revealed a slightly increased corneal diameter, abnormal flattening of the endothelium, numerous iris processes and fibrous strands in the angles, normal trabeculum and canal of Schlemm, absence of normal iris crypts, a thin endothelial membrane on the anterior surface, scattered infiltration of small round cells, and beginning ectropion uveas. The ciliary body was small, flat, associated with deficient circular muscle fibers and attenuated processes, which were displaced backward. The lens was small and spheroid in shape. The outer fibers showed fragmentation and scattered vacuoles with early calcification. Both lenses were dislocated.

This case will be discussed in detail with special emphasis upon pathologic changes which will be shown with lantern slides. Complications associated with trauma, such as dislocation of lens, glaucoma and separation of retina, will be discussed. Technical difficulties of cataract extraction, and retinal reattachment in these cases will be discussed. The importance of the presence of atypical cases of arachnodactyly with possible ocular complications will be stressed.

9:10 a.m.

A STUDY OF THE ROLE OF TOXOPLASMOSIS IN ADULT CHORIORETINITIS

Alan C. Woods, M.D.
Baltimore, Md.
and
Leon Jacobs, Ph. D
BY INVITATION
Ronald M. Woods, Ph. D.
BY INVITATION
M. K. Cook
BY INVITATION
Bethesda, Md.

Discussers:
Michael J. Hogan, M.D.
San Francisco, Calif.
and
Dr. Norman H. Ashton
London, England
BY INVITATION

This is a statistical study of the results of dye tests in 98 nonuveitis controls, 110 cases on nongranulomatous uveitis, and 201 cases of granulomatous uveitis. The incidence of significantly positive dye tests in the first two groups was 25 per cent, and in the granulomatous group was 45 per cent. The average titers were much higher in the granulomatous group. When these positive dye-test results in the granulomatous uveitis group were studied according to their disposition in the various etiologic subgroups, it was found the greatest number of such positive tests fell in the large group in which the previous medical survey had revealed no etiologic factor to which the uveitis could be attributed. The histories of all patients with granulomatous uveitis giving positive dye tests were then re-examined to determine if, in the light of the

positive dye test, the original diagnosis should be sustained or revised. In 10 cases a diagnosis of the congenital toxoplasmosis had already been made. In 113 cases attributed to conventional etiologies, there were 33 positive dye tests, usually of low titers. On review, it appeared that in 6 of these a diagnosis of ocular toxoplasmosis should be considered. In 27 the original diagnosis was sustained. In 78 cases of undetermined etiology, there were 44 positive dye tests, and in 42 of these the diagonsis of ocular toxoplasmosis appeared justified.

In the entire series there were 58 cases of possible or probable ocular toxoplasmosis—17 of congenital toxoplasmosis, and 41 of apparently acquired infection, an incidence of 8 and 20 per cent.

The usual ocular picture was a "focal" choroiditis (41 cases), a disseminated choroiditis (2 cases), a generalized granulomatous uveitis (10 cases), and a retinal periphlebitis with vitreous hemorrhage (5 cases).

It was concluded, accepting the premises that the bodies described by Wilder were true Toxoplasma parasites and that the titers used in this study were indicative of a present or past Toxoplasma infection, that this study indicated that adult acquired toxoplasmosis was an actual clinical entity. The criteria on which such a diagnosis could be made are outlined.

9:50 a.m.

SEROLOGIC DATA ON ADULTS WITH HISTOLOGICALLY DIAGNOSED TOXOPLASMIC CHORIORETINITIS

Helenor Campbell Wilder
Washington, D.C.
and
Leon Jacobs, Ph.D.
BY INVITATION
M. Katherine Cook
BY INVITATION
Bethesda, Md.

Serologic tests for toxoplasmosis were performed on 20 adults in whose ocular lesions Toxoplasma-like protozoa were found on microscopic examination. The Sabin-Feldman dye test was positive to some degree in every instance: 2 positive undiluted; 1 positive 1:2; 8 positive 1:16; 3 positive 1:32; 4 positive 1:64; 1 positive 1:256; 1 positive 1:2048. The implications of these results are discussed in relation to length of time between enucleation and serologic test, and to age of patient when tested.

10:15 a.m.

A SIMPLIFIED DACRYOCYSTORHINOSTOMY (A Motion Picture)

Charles E. Hiff, M.D.
Baltimore, Md.

The external method of dacryocystorhinostomy has been greatly simplified by use of the Stryker saw and nasolacrimal catheter.

The Stryker saw has an oscillating rather than rotary action and cuts only bone without endangering the soft tissues. The special ten millimeter trephine for dacryocystorhinostomy has been designed to cut an adequate nasal window cleanly, and of sufficient size, to minimize the possibility of late closure.

The use of a nasolacrimal catheter eliminates the difficult procedure of suturing nasal to sac mucous membrane and produces an excellent tract for epithelization.

Results have been highly satisfactory.

10:30 a.m.—Recess

10:35 a.m.

CONTACT LENSES: AN EVALUATING STUDY

James L. McGraw, M.D.
Syracuse, N.Y.
and
Jay M. Enoch, M.D.
BY INVITATION
Fort Knox, Ky.

Discussers:

Arno E. Town, M.D.
Philadelphia, Pa

George K. Smelser, Ph. D.
New York, N. Y.

An intensive comparative study of four contact lenses and spectacles was carried out at the Army Medical Research Laboratory at Fort Knox, Kentucky, to determine the relative merit of each and thence determine the possible application to the Armed Forces.

Laboratory studies included subjective and objective measurements of corneal turbidity, halo brightness and time of onset halos, entoptic field changes, color vision changes, visual acuity and tolerance to light at different levels of target and surround illumination, depth perception, binocular vision and fusion tests, dark adaptation, and clinical appearance of the eye. Field studies were conducted in an effort to simulate all conditions that a military man might encounter in routine life and in the field of battle.

The comparative results of the study are largely demonstrated on a series of graphs. Corneal haze was found to develop only with wearing of the fluid lens; therefore, most of the changes demonstrated were with this lens.

The two fluidless ventilated corneal-scleral lenses proved far superior in performance. Asymptomatic wearing time of fourteen to eighteen hours was the rule. No corneal changes were demonstrated and therefore visual acuity, tolerance to light, etc., were unaffected.

The performance of contact lenses as compared with spectacles was frequently superior, especially in field work and in inclement weather. There are few advantages in routine office duty.

Recommended studies are toward more simplified fitting technics and improvement in plastics in an effort to eliminate solutions such as wetting agents completely.

11:10 a.m.

DIFFERENTIATION AND TREATMENT OF ECZEMAS OF THE EYELIDS

Frederick H. Theodore, M.D.
New York, N.Y.

Discussers:
Phillips Thygeson, M.D.
San Jose, Calif.
Alson E. Braley, M.D.
Iowa City, Iowa

The successful management of eyelid eczemas requires differentiation of the major causes of the condition, as the treatment will vary accordingly. An incorrect diagnosis leading to faulty therapy not only will yield no benefit but may result in aggravation of the original dermatitis. In practice three main groups are encountered: (1) allergic eczematous dermatitis, due to contact allergy, in which the basic treatment is elimination of the allergens; (2) infectious eczematoid staphylococcal dermatitis secondary to eyelid infection, the most common cause of chronic eczema encountered by ophthalmologists, in which intensive treatment with anti-infective medicaments and staphylococcus toxoid is indicated; and (3) certain generalized dermatoses such as atopic dermatitis, seborrhea and psoriasis, in which the nonspecific eyelid eczema, that occurs along with diagnostic lesions elsewhere on the skin, requires gentle, stage-by-stage, symptomatic treatment. Differentiation of these clinical entities is of great value in the treatment of eyelid eczemas, which often may present therapeutic problems. Many of these difficulties may be avoided if such patients are seen by the ophthalmologist to determine if external ocular disease plays a role in the pathologic process.

SECTION ON OTOLARYNGOLOGY

Tuesday, October 13, 1953
2:00 p.m.
GRAND BALL ROOM

Presiding Officer:
HENRY B. ORTON, M.D.
Vice-President

Secretary:
HOWARD P. HOUSE, M.D.

SYMPOSIUM: THE PROBLEM OF NOISE IN INDUSTRY

(Sponsored by the Sub-Committee on Noise in Industry of the Committee on Conservation of Hearing)

The Problem of Hearing in Industry — A History of Recent Years

Gordon D. Hoople, M.D.
Syracuse, N.Y.

The problems of noise have multiplied greatly with modern industrial methods.

A historical review of this problem will be presented for the purpose of acquainting the otologist with his responsibilities in this sphere of activity.

Report of the Field Representative

Douglas Wheeler, Ph. D.
Los Angeles, Calif.
BY INVITATION

This will be a summary of the types of industrial situations studied, together with some findings resulting from these studies to date. Data will be presented on hearing losses presumably resulting from noise exposure, survey data descriptive of the studies of hearing acuity in the contemporary industrial plant, and some explanatory data on the performance of ear protectors. The probable course of future industrial noise research will be outlined.

Medico-Legal Aspects

C. Stewart Nash, M.D.
Rochester, N.Y.

Noise in industry entails problems in compensation and liability. In fairness to labor, industry and the public economy, the otological consultant in occupational deafness should understand the rights and responsibilities of each of these groups to the others. In addition he should know the methods and procedures involved in these problems along with their application and limitations.

Testing Procedures in the Office

Aram Glorig, M.D.
Washington, D.C.

The problem of noise in industry, and its effect on hearing, has added a new responsibility to the work of the otolaryngologist, and particularly the otologist. It is my purpose to enlarge upon this, pointing out some of the many facets of the growing and important role of the otolaryngologist in the industrial noise program. Conservation of hearing is the ultimate goal of the industrial noise program, and this is, after all, primarily a problem for the otologist.

2:45 p.m.

SYMPOSIUM: TINNITUS—NEW CONCEPTS IN ETIOLOGY AND MANAGEMENT

Anatomic Aspects

Benjamin Spector, M.D.
Boston, Mass.
BY INVITATION

The neuro-anatomic pathways involved in the auditory radiations and the influence of the autonomic nervous system on subjective sensory phenomena will be reviewed. The deviations from the norm will be stressed in regard to participation of the central nervous system.

Physiologic Aspects

Hallowell Davis, M.D.
St. Louis, Mo.
BY INVITATION

Recent physiologic developments in cochlear dynamics will be presented. The participation of the organ of Corti, stria vascularis, and the first order of neurons will be reviewed in their potential participation in the etiology of cochlear tinnitus. The recent demonstration of resting cochlear potentials will be analyzed in the light of potential causes of certain types of tinnitus. Neurophysiologic facts pertaining to episodic tinnitus will be covered.

Otologic Aspects

Victor Goodhill, M.D.
Los Angeles, Calif.

The otologic problem of tinnitus as a whole will be reviewed. Significant otologic diseases characterized by profound tinnitus will be discussed. The management of the patient who has tinnitus, as a psychosomatic entity, will be stressed. A brief review of current methods of treatment of the symptom of tinnitus will be presented.

3:45 p.m.

SYMPOSIUM: MEDICAL MANAGEMENT OF CHRONIC OTIC SKIN INFECTIONS

Moderator

Francis W. Davison, M. D.
Danville, Pa.

Rationale of Therapy

Ben H. Senturia, M.D.
St. Louis, Mo.

A review of the known physiologic and biochemical mechanisms of skin, which contribute to the resistance of the ear canal against contamination and infection, is planned. The histology and pathology of the skin of the external auditory canal will be reviewed. Biopsies obtained prior to and after skin grafting of mastoid and fenestration cavities will be compared. Familiarity with the normal and pathologic skin functions will lead to a better understanding of the pathogenesis of diseases of the ear. With this knowledge in hand we can more effectively combat underlying mechanisms as well as the superimposed bacterial and mycotic diseases which infest the ear canal, postoperative mastoid and fenestration cavities. The rationale of treatment based on these considerations will be presented.

Laboratory Diagnostic Technics

H. Russell Fisher, M.D.
Los Angeles, Calif.

BY INVITATION

The traditional use of bacteriologic examination of the aural exudate to reveal the presenting bacterial flora has been expanded into a therapeutic guide by the application of antibiotic sensitivity testing. Selection of an antibiotic or antibacterial agent on the basis of individual bacterial sensitivity tests has favored the prompt application of a potentially effective therapeutic substance, replacing empirical "trial and error" or "shotgun" prescribing. The results of the use of specific therapy in a relatively large series of ear cases are reported.

In an effort to expedite specific therapy, save tissue destruction and shorten the period of morbidity, routine laboratory methods have been modified to favor a "therapeutic diagnosis" within twenty-four hours. These methods and adaptations of procedure to various clinical situations, particularly those encountered in office practice, are described in detail.

Therapeutic Results

Edmund P. Fowler, Jr., M.D.
New York, N.Y.

Clinically there are many factors which contribute to the alleviation of chronic external otitis and to the drying up of postoperative radical cavities. These include the presence of foreign bodies, the thoroughness of the surgery, the size of the meatal opening, the number of times per week the area is cleansed, the season of the year, the introduction of water into the ear, psychic factors and, last but not least, the type of infection and the susceptibility to drugs. The apparent relative value of different medications in various media will be analyzed, according to experiences in New York, St. Louis and Los Angeles.

4:30 p.m.

A SATISFACTORY METHOD OF TREATING SECRETORY OTITIS

Aubrey G. Rawlins, M.D.
San Francisco, Calif.

Discussers:

Gordon D. Hoople, M.D.
Syracuse, N.Y.

Oliver W. Suehs, M.D.
Austin, Texas

Secretory otitis is a pathologic condition in which the eustachian tube has lost some of its normal functions. This malfunctioning results in a collection of fluid in the middle ear and its adnexa.

There are many causes for eustachian tube malfunction and each will be discussed in detail. A clinical classification based upon the type of fluid present in the middle ear will be presented.

A satisfactory treatment will be outlined and presented in detail.

SECTION ON OPHTHALMOLOGY

Wednesday, October 14, 1953
9:00 a.m.
GRAND BALL ROOM

Presiding Officer:
FREDERICK C. CORDES, M.D.
President

Secretary:
KENNETH L. KOPER, M.D.

CYCLOPIA
(A Clinicopathologic Case Report)

Windsor S. Davies, M.D.
Detroit, Mich.

An 18-year-old white primipara delivered a live male infant which expired one hour after birth. The infant showed a cyclops deformity with characteristic deformation of the skull and face. There were no malformations of any other portion of the body. The mother's history during pregnancy revealed no evidence of infection, malnutrition or trauma.

9:05 a.m.

SYMPOSIUM: CATARACT EXTRACTION

Moderator
Daniel B. Kirby, M.D.
New York, N.Y.

Preparation of the Patient

Walter S. Atkinson, M.D.
Watertown, N.Y.

Incision and Closures; Sutures
Iridectomy and Iridotomy
Principles of Wound Healing

Frederick A. Davis, M.D.
Madison, Wis.

Extracapsular Extraction,
Planned and Unplanned

Wilfred E. Fry, M.D.
Philadelphia, Pa.

Intracapsular Extraction:
Mechanics, Technics

Derrick Vail, M.D.
Chicago, Ill.

Complicated Cases

John M. McLean, M.D.
New York, N.Y.

Complications Occurring During Surgery

William B. Clark, M.D.
New Orleans, La.

Complications After Extraction
A. Clinical Aspects

Paul A. Chandler, M.D.
Boston, Mass.

B. Pathologic Aspects

Jack S. Guyton, M.D.
Baltimore, Md.

Summary

Daniel B. Kirby, M.D.
New York, N.Y.

SECTION ON OTOLARYNGOLOGY

Wednesday, October 14, 1953
2:00 p.m.
GRAND BALL ROOM

Presiding Officer:
THOMAS C. GALLOWAY, M.D.
Vice-President

Secretary:
HOWARD P. HOUSE, M.D.

PRESENTATION OF NEW INSTRUMENTS AND NEW TECHNICS

2:10 p.m.

SYMPOSIUM: CARCINOMA OF THE LARYNX

Moderator
Gordon B. New, M.D.
Rochester, Minn.

Diagnosis

A. C. Furstenberg, M. D.
Ann Arbor, Mich.

The introduction of this symposium will emphasize the importance of a careful evaluation of the clinical behavior of the lesion under consideration. The danger of accepting a pathologic diagnosis in every instance without due regard to the clinical appearance of the lesion, and its progress, will be stressed. Errors of omission and commission may be reduced to the minimum by giving due

recognition to a careful appraisal of all factors derived from a thorough clinical study of malignant disease in conjunction with laboratory findings.

Consideration of Transoral Removal

Francis LeJeune, M.D.
New Orleans, La.

Intrinsic carcinoma of the vocal cords is occasionally seen as a small growth on one vocal cord. This growth, if seen early, is no larger than the head of a match or a small green pea with visible normal tissue on all sides. Such growths are similar to surface lesions and may be completely and sucessfully removed by intralaryngeal surgery.

Laryngofissure

Chevalier Jackson, M.D.
Philadelphia, Pa.

The indications and contraindications for laryngofissure surgery will be presented in detail. A modification of the classical laryngofissure procedure will be discussed.

Evaluation of Hemilaryngectomy with Immediate Skin Graft

Frederick A. Figi, M.D.
Rochester, Minn.

There are definite limits to the extent of tissue that can be removed by means of thyrotomy or by classical hemilaryngectomy without destroying function or endangering collapse of the larynx. Relining this structure with a skin graft at times has been carried out secondarily to correct the stenosis induced by exceeding these limits. Hemilaryngectomy with immediate skin grafting renders more extensive local removal possible without disturbing normal physiology. The procedure has now been performed for a sufficient number of patients to permit adequate evaluation, and it seems certain to be applied more frequently in the future, since in a group of fairly advanced carcinomas it obviates the necessity for total laryngectomy. The procedure is feasible when fixation is present anteriorly, and is particularly useful for the removal of moderately active and low-grade malignant lesions. The results after initiation of this operation more than five years ago have been highly satisfactory; the airway has been free in all cases, the voice has ranged from very husky to almost normal, and in none of the patients has there been a recurrence of the neoplasm.

Evaluation of Total Laryngectomy

LeRoy A. Schall, M.D.
Boston, Mass.

Between 1931 and 1953, 306 patients with cancer of the larynx have been treated by the removal of the larynx, either by myself or by my associates operating under my direction and using my technic. Of these 306 patients, 165 were operated on between the years of 1931 and 1948; hence, five years have elapsed since their surgery.

In keeping with the principles of cancer surgery, we have always performed a wide resection of the larynx. The technic of our procedure is described.

In evaluating the value of total laryngectomy for cancer of the larynx, our records were analyzed. All but two patients could be traced, and these two must be considered dead. Our results are:

Living and well 5 years or longer	97	
Dead after 5 years — no cancer	3	
5-year or longer survival	100	60.6%
Dead with local recurrence	39	23.6%
Operative mortality	2	1.2%
Dead with distant metastasis	8	4.8%
Unable to trace — presumed dead	2	1.2%
Dead in less than 5 years (other causes)	14	8.4%

Gland Dissection

Henry B. Orton, M.D.
Newark, N. J.

It is unnecessary to do prophylactic block dissection in intrinsic growth cordal cancer. I think it should be done in the epilaryngeal group including lesions of the aryepiglottic fold, ventricle, pyriform sinus and some of the low epiglottic lesions. In the subglottic lesions, a prophylactic block dissection may be necessary since the lymphatic drainage may go to either side. With lesions on the lateral pharyngeal wall, a prophylactic

block is done at the time of the pharyngotomy. If nodes are palpable at the time of operation from any of these lesions, a block dissection is in order.

Radiation

J. A. del Regato
Colorado Springs, Colo.
BY INVITATION

Squamous cell carcinomas which develop in the hypopharynx and endolarynx are biologically amenable to cure by irradiation.

Secondary characteristics, such as the location, the extent of infiltration, secondary infection, irreparability of affected tissues and the presence of metastases, often determine the radiocurability in a specific case.

Although some extensive inoperable lesions may be controlled by radiotherapy, carcinomas with the most favorable outlook when treated by irradiation are the superficial, small and not infiltrating lesions which are most often found in the endolarynx and which may also be successfully treated by surgery.

The outstanding asset of radiotherapy is its conservatism. This conservatism is enhanced and the complications minimized by the use of high voltage good quality radiation and protraction of the treatment over several weeks.

Summary

Louis H. Clerf, M.D.
Philadelphia, Pa.

SECTION ON OPHTHALMOLOGY

Thursday, October 15, 1953
9:00 a.m.
GRAND BALL ROOM

Presiding Officer:
HARVEY E. THORPE, M.D.
Vice-President

Secretary:
KENNETH L. ROPER, M.D.

TORULA UVEITIS
(A Clinicopathologic Case Report)

F. Phinizy Calhoun, Jr., M.D.
Atlanta, Ga.
and
Harold E. Wager, M.D.
Panama City, Fla.

A 40-year-old woman developed a low grade fever, boring headaches, and photophobia, which progressed to mental apathy and neck rigidity. Spinal puncture revealed a turbid, tenacious spinal fluid greatly increased in pressure. Smear of the fluid showed a yeast-like organism which upon culture proved to be Cryptococcus neoformans. During the next two months there were four exacerbations of the condition, each one treated by antibiotics, sulfa drugs and spinal taps.

Approximately nine weeks after onset of the general illness the left eye became inflamed and the vision blurred. The first ocular examination was performed two weeks later and revealed the vision to be OD 20/20 and OS 20/30. The right eye was normal in all respects. The left eye showed a marked anterior uveitis with dense posterior synechias. No details of the fundus could be made out. Despite extensive local treatment, the eye condition became worse, and the eye was removed approximately two months after onset of the eye condition. The patient died two months later. Autopsy was not obtained.

Microscopic examination revealed a large localized gelatinous inflammatory mass in the lower temporal quadrant of the choroid which was the source of a severe endophthalmitis. Round yeast organisms, many of them showing the typical capsule, were found in the subretinal exudate.

There are only a very few reports in the literature of involvement of the eye in human torulosis.

9:10 a.m.

PRESENTATION OF NEW METHODS AND NEW INSTRUMENTS

9:25 a.m.

CLINICAL DETECTION OF THE CARRIER STATE IN OPHTHALMOLOGIC PATHOLOGY
(Jackson Memorial Lecture)

Harold F. Falls, M.D.
Ann Arbor, Mich.

9:55 a.m.

ESCLERECTOMIA LAMELAR EN EL DESPRENDIMIENTO DE LA RETINA (LAMELLAR SCLERECTOMY IN RETINAL DETACHMENT) (A Motion Picture)

Olga M. Ferrer, M.D.
Havana, Cuba
BY INVITATION

10:05 a.m.

ANTIHYALURONIDASE AND ANTISTREPTOLYSIN TITERS IN UVEITIS

Irving H. Leopold, M.D.
and
Thomas G. Dickinson, M.D.
BY INVITATION
Philadelphia, Pa.

Discussers:

Alan C. Woods, M.D.
Baltimore, Md.

Frank W. Newell, M.D.
Chicago, Ill.

The study of the etiology of uveitis has long presented one of the most vexing problems with which the ophthalmologist has had to contend. Inflammatory conditions of the uveal tract can be classified generally into three etiologic groups: those caused by injury, those caused by direct extension of inflammatory conditions of other parts of the eye and those which are considered to be caused by endogenous infections and their sequelae.

Woods has further classified this third group into two main subdivisions; namely, granulomatous and nongranulomatous. Of the granulomatous uveitis, the major causes are thought to be syphilis, tuberculosis, sarcoid, brucellosis, toxoplasmosis, various fungous infections, nematode infestations and virus diseases. In theses there has been occasional evidence of actual invasion of the uvea by the causative organism. In the nongranulomatous uveitis, the cause is thought to be a sterile allergic reaction or a hypersensitivity of the ocular tissues to a specific bacterium or its products. The hemolytic streptococcus and the gonococcus have been suggested as the principal offenders.

It is characteristic of the growing hemolytic streptococcus to liberate into the surrounding tissue a number of independently acting extracellular substances. The four most important of these are: (1) streptococcal fibrinolysin or streptokinase, (2) streptococcal desoxyribose nuclease or streptodornase, (3) streptococcal hemolysin or streptolysin, (4) streptococcal hyaluronidase.

These four extracellular substances liberated by the hemolytic streptococcus plus two fractional parts of the streptococcal cell; namely, the cytoplasmic particles and the supernate or nuclear fraction, being protein in nature, can act as antigens and produce specific antibodies in the blood stream. These antibodies in the blood stream produced as a consequence of widely prevalent human contact or infection with the streptococcus have a characteristic age distribution. The antihyaluronidase and antistreptolysin titers were measured in a random series of cases of uveitis in order to learn if any apparent relationship could be demonstrated between this disease and streptococcal exotoxins in the body. Sixty-three cases were chosen from the ward services of the Wills Eye Hospital. Statistical analyses applied to the results suggest that the cases of uveitis in this study do not represent a normal group with regard to serum antihyaluronidase and antistreptolysin titers. These data suggest that the hemolytic streptococcus may play an etiologic role in the production of uveitis. Also, these data suggest that these tests, particularly the antihyaluronidase titers, should be studied further as a possible method of detecting the etiology in cases of uveitis.

10:45 a.m.—Recess

10:50 a.m.

ROENTGEN DIAGNOSIS OF LACRIMAL GLAND TUMORS

Raymond L. Pfeiffer, M.D.
Ira S. Jones, M.D.
New York, N.Y.

This study describes the features in orbital roentgenograms which call attention to the lacrimal gland. Emphasis is placed upon frequent and important contributions which x-rays make in the diagnosis of lacrimal gland tumors. The value of the roentgen appearance in differentiating various types of tumors of the gland is also discussed. Particular attention is given to a delineation of the prog-

nostic value of x-rays both in aiding the surgeon to decide upon the proper treatment and also in following the subsequent course of the disease.

11:05 a.m.

LACRIMAL GLAND TUMORS: PROGNOSIS AS DETERMINED BY PATHOLOGY

Arnold W. Forrest, M.D.
White Plains, N.Y.
BY INVITATION

Discussers:

Vincent C. Johnson, M.D.
Detroit, Mich.
BY INVITATION

Theodore E. Sanders, M.D.
St. Louis, Mo.

This study of lacrimal gland tumors demonstrates that the mixed tumor comprises several distinct types. Some types lead to early death of the patient while other types are compatible with survival for several decades without evidence of disease. The diagnostic criteria for the various types are shown and their behavior is documented.

11:40 a.m.

CHORIORETINAL BURNS PRODUCED BY ATOMIC FLASH

Victor A. Byrnes, M.D.
and
David V. L. Brown, M.D.
BY INVITATION
Heinrich W. Rose, M.D.
BY INVITATION
Paul A. Cibis, M.D.
BY INVITATION
Randolph Field, Texas

Discusser:

Jonas S. Friedenwald, M.D.
Baltimore, Md.

Atomic explosions present a real threat of eye damage through high intensity light. The damage produced by blast, gamma radiation, and neutrons has been extensively reported, but intraocular damage produced by infrared and visible light rays has not been adequately studied.

The amount of radiant energy required to produce thermal damage to the choroid and retina in pigmented eyes has been experimentally determined in the laboratory. Following this, rabbits were exposed during atomic bomb detonations. The retinal burns produced are typical and follow the predictions based on calculations. Kodachrome fundus photographs show these lesions well.

Four human cases are included in the series. Lesions present clinically resemble those seen in the rabbit. When exposures occur at night with a widely dilated pupil the lesions can be severe if the individual is near the detonation. Visible lesions are permanent and produce scotomas equivalent to the damaged area. In some cases the nerve fiber layer is not destroyed and in those cases sector defects are not formed. The blink reflex is too slow to be of protective value because of the extremely high energy output in the first 0.1 second after detonation of the bomb.

The histologic sections show that the greatest energy absorption lies, as predicted, in the pigment layer of the retina and choroid. This is therefore the area of greatest damage, and proper terminology for the condition is chorioretinal burn.

SECTION ON OTOLARYNGOLOGY

Thursday, October 15, 1953
2:00 p.m.
GRAND BALL ROOM

Presiding Officer:
HENRY B. ORTON, M.D.
Vice-President

Secretary:
HOWARD P. HOUSE, M.D.

SOME CONTRIBUTIONS OF LABORATORY RESEARCH TO THE DEVELOPMENT OF FENESTRATION SURGERY
(The Wherry Memorial Lecture)

George E. Shambaugh, Jr., M.D.
Chicago, Ill.

2:30 p.m.

STUDY OF LIVING CELLS TAKEN FROM THE HUMAN EYE, NOSE AND THROAT
(A Motion Picture)

(Sponsored by the American Society of Ophthalmologic and Otolaryngologic Allergy)

Charles M. Pomerat, Ph. D.
Galveston, Texas
BY INVITATION

Relatively simple methods are now available which make possible the cultivation of a wide variety of adult human tissues. Both normal and pathological materials can thus be studied under highly controllable conditions.

The tissue culture medium usually employed consists of avian plasma and embryonic extract, generally enriched with human placental cord serum or ascitic fluid derived from patients with malignancies. The double cover slip and roller tube methods are recommended. Fragments of tissues 1 to 2 mm. square embedded is a plasma clot usually show cellular emigration in from two to four days except in highly proliferative young, normal tissues and some malignancies. Anaplastic tumors occasionally exhibit cellular outgrowth within a few hours.

Epithelial sheets have been produced from cornea and conjunctiva as well as from the nasal and pharyngeal mucosae. Spindle cells presumably fibroblasts readily emigrate from the stromal tissues. Macrophages together with various blood elements are common. Occasionally endothelial sprouts are produced.

Cultures of tissues from the eye, nose and throat have been photographed both with still and moving picture technic. The use of phase contrast optics reveals cell organoids such as mitochondria which are of very great interest in view of the fact that many enzymes are known to be associated with these bodies. By photographing the cultures with a time-lapse device, the activity of cells can be accelerated so that the projection of resulting films reveals behavioral features heretofore unknown for tissues which have been studied for many years with the technics of "fixed" histology. Thus, it has been demonstrated that the nuclei of cells from the human nasal mucosa show extraordinary rotatory activity.

By placing the cultures in a perfusion chamber, it is not only possible to observe the activities of groups of cells or parts of individual cells but also their reaction to the introduction of special nutrients, drugs and antigens. Many suggestions for fresh research attacks emerge from the methods available in the study of tissues from the eye, nose and throat.

3:10 p.m.

SYMPOSIUM: MEDICAL MANAGEMENT OF DIZZINESS

The Importance of Accurate Diagnosis

David D. DeWeese, M.D.
Portland, Ore.

Dizziness is one of the most common and most confusing symptoms presented by the patient. A carefully taken history is important in evaluation of this symptom. It is also important that whirling vertigo be distinguished from other sensations described as dizziness by the patient.

Systematized vertigo usually indicates disease of the statokinetic system. Nonsystematized vertigo may arise from disease in any part of the body. Disease of the proprioceptive, cardiovascular and central nervous systems commonly gives rise to dizziness outside the statokinetic system.

Management will often be unsuccessful if all paroxysmal dizziness is loosely called Meniere's syndrome. Incomplete study of the patient will lead to a high percentage of incorrect diagnoses of functional dizziness. Proper diagnosis will lead to proper management in a high percentage of cases.

Certain patterns must constantly be borne in mind. The otolaryngologist should become increasingly more alert to general systemic diseases in which dizziness is one symptom.

The Internist's Approach

Laurence Selling, M.D.
Portland, Ore.
BY INVITATION

Treatment of dizziness is determined by its cause. For this discussion, it can be divided into four general groups: 1. Dizziness stemming from impairment of the peripheral structures 2. Dizziness associated with lesions of the central nervous system 3. Dizziness caused by other systemic diseases 4. Dizziness on a func-

tional basis, chiefly the anxiety tension states.

Dizziness stemming from impairment of the peripheral structures can be divided again, grossly, into (a) Meniere's syndrome and (b) toxic labyrinthitis. The otolaryngologist has had experience with the Meniere group. While many basic principles of therapy are well know, there are still controversial methods. Because patients fear what their dizziness may mean, it is important to explain to them what the labyrinth is and how it can be upset by toxemia or infection.

Dizziness associated with lesions of the central nervous system is only part of the larger picture. Its treatment depends on the underlying factor.

Dizziness may be one symptom of such systemic diseases as hypoglycemia, allergy, hypertensive cardiovascular disease and epilepsy. Proper management of the underlying disease will frequently relieve the dizziness. To accomplish this, cooperation of the internist and otolaryngologist is necessary.

Dizziness on a functional basis is probably most common. In treating this dizziness the internist, I think, has a different approach than the otolaryngologist. Many of these patients can be helped by relatively simple methods.

Pharmacology

Elton L. McCawley, Ph. D.
Portland, Ore.
BY INVITATION

Successful therapy of vertigo or dizziness depends on an accurate diagnosis and elimination of causative factors. Where such causative factors are unknown or impossible to treat, symptomatic medical management of dizziness is disappointing.

The use of histamine, nicotinic acid, riboflavin and vitamin B complex are not always effective, and reduction of alleged vasospasm is unlikely. Reduction of body sodium and elevation of potassium may be effective but is difficult to obtain and manage.

The recent introduction of dimenhydrinate (Dramamine) has been encouraging, provided high enough dosage is used. Further advances in this field with other agents are expected. The neuropharmacology and locus of action of dimenhydrinate in the reduction of the objective sign of nystagmus are discussed. Avenues of future progress and need for clinical evaluation of therapeutic procedures are indicated.

4:10 p.m.

AN ANALYSIS OF SINUS SURGERY

W. Likely Simpson, M.D.
Memphis, Tenn.

Discussers:

G. L. Whelan, M.D.
Philadelphia, Pa.

Benjamin J. Shuster, M.D.
Philadelphia, Pa.

An analysis of the operative procedures performed on 324 external-ethmo-sphenofrontal sinuses on 200 patients, done over a period of thirty years, will be presented. Indications, technic, pathologic findings, and complications will be discussed.

SECTION ON OPHTHALMOLOGY

Friday, October 16, 1953
9:00 a.m.
GRAND BALL ROOM

Presiding Officer:
FREDERICK C. CORDES, M.D.
President

Secretary:
KENNETH L. ROPER, M.D.

ORBITAL CHOLESTEATOMA
(A Clinicopathologic Case Report)

Lawrence R. Dame, M.D.
Greenfield, Mass.

Benjamin Alport, M.D.
Springfield, Mass.

An isolated tumor may be a cholesteatoma arising by hyperplasia of congenitally or traumatically misplaced epithelial cells. There is an increased cholesterol content. Erosion of bony structures is possible. Occurrence in the orbit is rare.

A 45-year-old white male caretaker had increasing discomfort for six months, di-

plopia and upper lid deformity with orbital bone erosion demonstrable by roentgen examination. All laboratory and physical findings were otherwise within normal limits. Operation with removal of the tumor resulted in a complete return to normal ocular physiology. Pathologic examination of the specimen revealed a cholesteatoma of the orbital bones.

9:10 a.m.

PRESENTATION OF NEW METHODS AND NEW INSTRUMENTS

9:25 a.m.

THE ROLE OF VITREOUS DETACHMENT IN APHAKIC AND MALIGNANT GLAUCOMA

Robert N. Shaffer, M.D.
San Francisco, Calif.

Discussers:

Peter C. Kronfeld, M.D.
Chicago, Ill.

Paul A. Chandler, M.D.
Boston, Mass.

Modern slit lamps and the Hruby lens have made the study of vitreous detachments possible. The role of these detachments in the production of retinal detachment has become common knowledge. Their role in glaucoma has received less attention.

In aphakia and in dislocated lenses, synechias can form between the iris and the vitreous face producing a pupillary block. Aqueous detaches the vitreous anteriorly or goes behind a posteriorly detached vitreous, shoving it forward producing a glaucoma. The anterior chamber can be either collapsed or filled with vitreous, depending on the site of the synechias and the type of vitreous detachment. Iridectomy will usually result in a cure of the glaucoma.

A similar syndrome is seen after lens extraction is done to relieve a malignant glaucoma. It becomes impossible to refill the anterior chamber because aqueous runs posteriorly forcing detached vitreous and iris forward, thus continuing the course of the malignant glaucoma.

Only by breaking the pupillary block can this type of glaucoma be relieved. If the aqueous is collecting anteriorly, iridectomy may be helpful. Usually aqueous collects behind the detached vitreous. The only chance of cure is actual incision through the detached vitreous to break the posterior anterior block.

10:05 a.m.

A STUDY OF PRIMARY AND AUXILIARY MEDIAL OCULAR ROTATION

Orville E. Gordon, M.D.
Chicago, Ill.

Discussers:

William E. Krewson, III, M.D.
Philadelphia, Pa.

Kenneth C. Swan, M.D.
Portland, Ore.

Medial ocular rotation has always been considered to be accomplished by unwrapping of the arc of contact between the medial rectus muscle and the eyeball. Measurement of the arcs of contact of the medial rectus muscle revealed that there was not enough arc of contact to permit the known full medial rotation.

The superior and inferior rectus muscles, whose secondary actions are internal rotation, were considered as the source of the medial rotation not supplied by the internal rectus. Experimental tenotomies were performed to see if this supposition had merit.

The recognition of an auxiliary means of internal rotation dependent on the vertical recti may help us anticipate the vertical tropias that occur after squint surgery on the horizontal recti.

10:45 a.m.—Recess

10:50 a.m.

STERILIZATION OF SHARP INSTRUMENTS BY BOILING, WITH UTILIZATION OF CATHODIC PROTECTION TO PREVENT CORROSION

William H. Havener, M.D.
C. A. Siebert, Ph. D.
Ann Arbor, Mich.

BY INVITATION

Discusser:

M. Hayward Post, M.D.
St. Louis, Mo.

Data is presented showing that moist heat at 100° C. is a more effective means of sterilization than is immersion in chem-

ical germicide. The use of the cathodic protection principle permits boiling of sharp instruments without damage to their edges. This principle is utilized by connecting a battery cathode to the instruments, and the anode to a sheet of metal immersed in the same solution (water). All corrosion in such a system is forced to occur at the anode, thereby protecting the cathodic instruments completely. A standard hospital sterilizer has been converted by the addition of a cathodic instrument tray and anodic metal plates and has been used successfully in the University of Michigan eye clinic for sterilizing sharp ophthalmic instruments.

11:20 a.m.

REPORT ON THE USE OF INTRAOCULAR ACRYLIC LENSES (RIDLEY OPERATION)

Warren S. Reese, M.D.
Philadelphia, Pa.
Hunter H. Romaine, M.D.
and
John Finlay, M.D.
BY INVITATION
New York, N. Y.

Discusser:

Selected Representative of the Cataract Symposium Panel

11:50 a.m.

SQUINT OPERATIONS AND BINOCULAR FUNCTION

Mr. Frank W. Law
London, England
BY INVITATION

Discusser:

Francis Heed Adler, M.D.
Philadelphia, Pa.

A series of cases of convergent concomitant strabismus are reviewed with regard to the factors indicating the need for operation and the type of operation adopted. Operative details are briefly indicated. The results in terms of degree of binocular function are assessed, and reviewed in the light of such factors as the age of onset of the squint, the binocular situation before operation, the interval between onset and operation, and the orthoptic treatment before and after operation.

SPECIAL SCIENTIFIC PROGRAMS

OTOSCLEROSIS STUDY GROUP
Division of
Committee on Conservation of Hearing

Private Dining Room 14
Saturday, 8:00 p.m.

JOHN R. LINDSAY, M.D.
President

Technic for Increasing Sound Differential Between Fenestra and Round Window to Enhance Hearing Improvement Following Successful Fenestration
George E. Shambaugh, Jr., M.D., Chicago, Ill.

Research Study on the Permeability of the Endolymphatic Labyrinth
Julius Lempert, M.D., New York, N. Y.

Circulation of the Spiral Ligament and Stria Vascularis in the Living Guinea Pig
Francis L. Weille, M.D., Boston, Mass.

TEACHERS' SECTION — OPHTHALMOLOGY

Crystal Room
Sunday, 10:00 a.m.

A. D. RUEDEMANN, M.D., *Chairman*

Visual Physiology: Trends in Ophthalmic Teaching

PANEL:

Arthur Linksz, M.D., New York, N. Y.
Alfred Cowan, M.D., Philadelphia, Pa.
Albert E. Sloane, M.D., Boston, Mass.
D. H. O'Rourke, M.D., Denver, Colo.
Gordon H. Gliddon, Ph.D., Hanover, N. H.
Hermann M. Burian, M.D., Iowa City, Iowa
Fred Jobe, Rochester, N. Y.

TEACHERS' SECTION — OTOLARYNGOLOGY

Private Dining Room 14
Sunday, 10:00 a.m.

LAWRENCE R. BOIES, M.D., *Chairman*

Objectives in Undergraduate Teaching
Francis L. Lederer, M.D., Chicago, Ill.

Transition from Specialization in Ear, Nose and Throat to Head and Neck Surgery
Joel J. Pressman, M.D., Los Angeles, Calif.

Comments on the Activities of the Teachers' Section from 1931 to 1942
Harry P. Schenck, M.D., Philadelphia, Pa.

COMMITTEE ON CONSERVATION OF HEARING
and
OTOSCLEROSIS STUDY GROUP DIVISION

Grand Ball Room
Sunday, 8:00 p.m.

DEAN M. LIERLE, M.D.
JOHN R. LINDSAY, M.D.
Co-Chairmen

Changes in Auditory Acuity Produced by Low and Medium Intensity Level Exposure
Scott N. Reger, Ph.D.
Dean M. Lierle, M.D.
Iowa City, Iowa

Blockage of the Round Window by Otosclerosis
John R. Lindsay, M.D.
William G. Hemenway, M.D.
Chicago, Ill.

Aids to Hearing
Kenneth M. Day, M.D., Pittsburgh, Pa.

INDUSTRIAL OPHTHALMOLOGY

Private Dining Room 14
Sunday, 8:00 p.m.

A. D. RUEDEMANN, M.D., *Chairman*
HEDWIG S. KUHN, M.D., *Secretary*

PANEL DISCUSSION
FIRST AID AND EMERGENCY CARE OF EYE INJURIES

Special Technics in Traumatic Eye Surgery
Edmund B. Spaeth, M.D., Philadelphia, Pa.

Bacterial Infections with Special Reference to Traumatic Eye Injuries
Col. J. Harry King, Jr. (MC), Washington, D. C.

Emergency and First Aid Technics As Practiced Inside Plant Gates
Ralph S. McLaughlin, M.D., Charleston, W. Va.

Some Special Items of Importance In Industrial Eye Care
Hedwig S. Kuhn, M.D., Hammond, Ind.

AMERICAN ORTHOPTIC COUNCIL
and
AMERICAN ASSOCIATION OF ORTHOPTIC TECHNICIANS

Private Dining Room 14
Tuesday, 8:00 p.m.

WALTER H. FINK, M.D.
President

FRANK D. COSTENBADER, M.D.
Secretary

SYMPOSIUM:
ACCOMMODATIVE ESOTROPIA

Physiology and Etiology
Marshall M. Parks, M.D., Washington, D. C.

Clinical Picture and Management
Frank D. Costenbader, M.D., Washington, D. C.

Orthoptic Treatment in the Office
Aleatha Tibbs, Washington, D. C.

Orthoptic Treatment at Home
Louisa Wells Kramer, Washington, D. C.

PLASTIC SURGERY

Grand Ball Room
Tuesday, 8:00 p.m.

JOHN MARQUIS CONVERSE, M.D.
WENDELL L. HUGHES, M.D.
Co-Chairmen

Lower Lid Repair
Sidney A. Fox, M.D., New York, N. Y.

Upper Lid Repair
Crowell Beard, M.D., San Jose, Calif.

Nerve Grafting for Repair of the Severed Peripheral Branches of the Facial Nerve
James H. Maxwell, M.D., Ann Arbor, Mich.

DISCUSSER:
Frank D. Lathrop, M.D., Boston, Mass.

The Deviated Nose and Septum
PANEL:
Oscar J. Becker, M.D., Chicago, Ill.
John Marquis Converse, M.D., New York, N. Y.
Edgar M. Holmes, M.D., Boston, Mass.
William C. Huffman, M.D., Iowa City, Iowa

MOTION PICTURES

Grand Ball Room
Thursday, 8:00 p.m.

HARVEY E. THORPE, M.D., *Chairman*
HOWARD P. HOUSE, M.D., *Secretary*

The Embryology of the Human Ear
Part I. The External and Middle Ear

COMMITTEE ON STUDY OF AUDIOVISUAL INSTRUCTION
Franz Altmann, M.D., *Consultant*
New York, N. Y.

Ocular Biomicroscopy

Milton L. Berliner, M.D.
Goodwin M. Breinin, M.D.
New York, N. Y.

Radical Neck Dissection

William C. Huffman, M.D.
Dean M. Lierle, M.D.
Iowa City, Iowa

Reconstruction of the Lacrimal Passageway After Trauma and Disease of the Lower Canaliculus

Alston Callahan, M.D.
Birmingham, Ala.

AMERICAN SOCIETY OF OPHTHALMOLOGIC and OTOLARYNGOLOGIC ALLERGY

Private Dining Room 14
Friday, 9:30 a.m.

KENNETH L. CRAFT, M.D.
President

MICHAEL H. BARONE, M.D.
Secretary

A New Diagnostic Aid in Allergic Deafness

C. W. Armstrong, M.D., Salina, Kan.

10:00 a.m.—Allergic and Other Untoward Reactions to Antibiotics and Drugs

French K. Hansel, M.D., St. Louis, Mo.

DISCUSSERS:

Leland H. Prewitt, M.D., Ottumwa, Iowa

Kenneth L. Craft, M.D., Indianapolis, Ind.

11:00 a.m.—Bacteriologic and Cytologic Criteria in Nasal and Sinus Disease

Joseph L. Goldman, M.D., New York, N. Y.

11:30 a.m.—Case Reports

Sinusitis—Allergy Plus Surgical Treatment

Sam H. Sanders, M.D., Memphis, Tenn.

Vasodilator Therapy for Nerve Type Deafness

George L. Loomis, M.D., Winona, Minn.

Psychogenic Allergy

Leland H. Prewitt, M.D., Ottumwa, Iowa

2:00 p.m.—Common Manifestations of Allergy in Ophthalmology

Albert N. Lemoine, Sr., M.D., Kansas City, Mo.

2:20 p.m.—Observations on Nontuberculous Phlyctenular Keratitis

Phillips Thygeson, M.D., San Jose, Calif.

2:40 p.m.—Use of Cortone and Chlor-trimeton Solution in the Treatment of Allergic Conjunctivitis

S. Albert Hanser, M.D., St. Louis, Mo.

3:00 p.m.—Therapeutics of Ocular Allergy

Irving Henry Leopold, M.D., Philadelphia, Pa.

SCIENTIFIC EXHIBITS

RED LACQUER ROOM
Kenneth L. Roper, M.D., *Chairman*
Chicago, Ill.

EXHIBIT S-1
TRENDS IN CAUSES OF BLINDNESS AMONG CHILDREN OF SCHOOL AGE

Exhibitor:
Franklin M. Foote, M.D.
BY INVITATION
National Society for the Prevention Blindness
New York, N. Y.

Co-Exhibitor:
Virginia Smith Boyce
BY INVITATION

This exhibit is based on studies of causes of blindness among children in schools for the blind made by the National Society during the past twenty years. Trends have been computed which indicate what has happened over the years to the etiologic factors such as injuries, infectious diseases, and hereditary factors. Material will be presented in the form of statistical charts and graphs with some of the important diseases illustrated by color photographs.

EXHIBIT S-2
PROBLEMS OF BIFOCAL LENSES

Exhibitor:
Ralph H. Pino, M.D.
Harper Hospital, Detroit, Mich.

Co-Exhibitor:
D. J. Edwards
BY INVITATION

The purpose of an optical system is to direct rays of light into determined pathways. This exhibit will point out ways and means of accomplishing this more adequately in bifocal, and especially aphakic lenses. Emphasis is placed on the reading point level, the position of the optic axis, and other aspects of mechanics that most effectively direct rays of light from the object through the optic axis of the lens, and on through the pupil to the macula.

EXHIBIT S-3
MIRROR LARYNGOSCOPY

Exhibitor:
Joseph I. Kemler, M.D.
Baltimore Eye, Ear and Throat Charity Hospital, and Sinai Hospitals
Baltimore, Md.

Co-Exhibitor:
Albert Steiner, M.D.

Laryngeal examinations and biopsy with the mirror in the indirect method are shown. Charts and x-ray films show the exact working in the larynx as well as operations and cauterizations in the larynx with the indirect method.

EXHIBIT S-4
ENDOTRACHEAL ANESTHESIA FOR TONSILLECTOMY AND ADENOIDECTOMY IN CHILDREN

Exhibitor:
O. Erik Hallberg, M.D.
Mayo Clinic, Rochester, Minn.

Co-Exhibitor:
John W. Pender, M.D.
BY INVITATION

The advantages of endotracheal anesthesia in adults for operation in or about the mouth have long been recognized but this method has not been used as widely in children. Six years of experience during which endotracheal anesthesia was used routinely for tonsillectomy and adenoidectomy in children has shown that this method leads to (1) more safety for the patient, (2) better exposure of the operating field for the surgeon and (3) more complete control of the anesthesia. The complications encountered in over a thousand children to whom endotracheal anesthesia was administered for tonsillectomy and adenoidectomy are presented. This exhibit demonstrates with models and diagrams the (1) position of the endotracheal tube in relation to the surgical instruments used during various

stages of the operation, and (2) the equipment used and the various methods employed to deliver the anesthetic gases through the endotracheal tube to the lungs.

EXHIBIT S-5
THE ARMED FORCES EYE FIELD CHEST

Exhibitor:
Col. J. H. King, Jr.
Ocular Research Unit,
Walter Reed Army Medical Center
Washington, D.C.
Co-Exhibitor:
Major John R. Fair
BY INVITATION

The Armed Forces Eye Field Chest is a set of surgical and diagnostic instruments for use in medical field installations. It contains many new developments in the way of folding perimeter, miniature slit lamp, and small electromagnet. The set supplements the basic surgical instrument set issued to field and evacuation hospitals.

EXHIBIT S-6
THE TEMPORAL BONE. AN ANATOMIC AND CLINICAL STUDY

Exhibitor:
J. Dudley Singleton, M.D.
Southwestern Medical School of the University of Texas, Dallas, Texas
Co-Exhibitors:
Louis E. Adin, Jr., M.D.
and
Ralph S. Clayton, M.D.
BY INVITATION

A correlation is shown between the different roentgen positions and the information to be derived from each, with drawings and specimens. A correlation between mastoid pneumatization and age is demonstrated. Specimens of temporal bones are used to demonstrate the different cell groups.

EXHIBIT S-7
ELECTRODIAGNOSIS OF LESIONS OF THE FACIAL NERVE

Exhibitor:
Emanuel K. Skolnik, M.D.
University of Illinois College of Medicine, Chicago, Ill.
Co-Exhibitor:
Arthur A. Rodriquez, M.D.
BY INVITATION

The exhibit is designed to illustrate the various methods of electrodiagnostic testing in lesions of the facial nerve. The three technics to be presented are (1) classical testing for reaction of degeneration utilizing the galvanic and faradic current; (2) strength frequency testing employing sine wave currents of varying frequencies, and (3) electromyography. Apparatus will be displayed and personnel will be available for discussion.

EXHIBIT S-8
ACCOMMODATIVE ESOTROPIA

Exhibitor:
Electra Healy, President
BY INVITATION
American Association of Orthoptic Technicians

The exhibit will endeavor to show the treatment of accommodative esotropia from the beginning to the eventual cure, step by step. Emphasis will be placed on the close cooperation of the ophthalmologist and the technician and how their observations can promote more effective treatment, with less effort by all concerned.

EXHIBIT S-9
RECRUITMENT TESTS AND THEIR CLINICAL APPLICATION

Exhibitor:
Raymond Carhart, Ph. D.
BY INVITATION
Department of Otolaryngology and School of Speech
Northwestern University, Chicago, Ill.
Co-Exhibitors:
George E. Shambaugh, Jr., M.D.
Eugene L. Derlacki, M.D.
and
James Jerger
BY INVITATION

The nature of the recruitment phenomenon and the method for demonstrating this abnormally rapid increase in loudness are briefly described. The more widely suggested tests for clinical evaluation of recruitment are reviewed. These tests include the alternate binaural loudness technic, measurement of difference limens for intensity change, and von Bekesy automatic audiometry. The performance on these tests of patients typi-

fying different clinical entities is summarized. Various interpretations of results, ranging from differential diagnosis of cochlear vs. neural lesions to the preoperative estimation of probable improvement from fenestration surgery, are surveyed, as are the comparative performances of the nerve deafened and the masked normal ear.

EXHIBIT S-10
AVIATION OPHTHALMOLOGY

Exhibitor:
Col. Victor A. Byrnes, (MC)
United States Air Forces School of Aviation Medicine
Randolph Field, Texas

Co-Exhibitor:
Capt. David V. L. Brown
BY INVITATION

The exhibit delineates changes in the atmosphere which occur with altitude and correlates these changes with function of the eyes. It points out factors of importance in air evacuation of patients. Some of the problems of aviation ophthalmology such as the importance of the lag in visual perception at high speeds are illustrated. Included also are new equipment items such as the Armed Forces Vision Tester and the high speed bail-out visor.

EXHIBIT S-11
HEARING LOSS IN CHILDREN WITH MULTIPLE HANDICAPS

Exhibitor:
Jane Shover, Associate Director
BY INVITATION
National Society for Crippled Children and Adults, Inc.
Chicago, Ill.

Technics of testing and treatment for use with children having multiple handicaps, will be shown by pictures, charts and demonstrations.

EXHIBIT S-12
DISSECTION IN CONTINUITY—THE COMMANDO OPERATION

Exhibitor:
Hans Von Leden, M.D.
Department of Otolaryngology
Northwestern University Medical School
Chicago, Ill.

The exhibit will include central moulage showing extent of operation and anatomic structures involved; colored drawings and legends to describe stages of procedure; preoperative and postoperative pictures of patients; and photomicrographs of primary lesions and involved lymphatics to show necessity for dissection in continuity.

EXHIBIT S-13
HEARING AID ANATOMY

Exhibitor:
Howard A. Carter
BY INVITATION
Council on Physical Medicine and Rehabilitation, American Medical Association
Chicago, Ill.

The physics of a hearing aid is presented by means of apparatus charts and demonstrational lectures. Comparisons are shown in the make-up, construction, and efficiency of operation of the vacuum tube vs. the transistor hearing aid.

EXHIBIT S-14
FUNGUS INFECTIONS OF THE UPPER RESPIRATORY AND ALIMENTARY TRACTS

Exhibitor:
Muriel Raum, M.D.
BY INVITATION
Armed Forces Institute of Pathology, Washington, D.C.

Co-Exhibitor:
Lt. Col. Lorenz E. Zimmerman, (MC)
BY INVITATION

Color transparencies will be used to demonstrate fungus infections and their tissue reactions involving areas of interest to the otolaryngologist. The pathologic material was obtained from cases in which the clinical symptoms were referable to a lesion accessible to biopsy by the otolaryngologist or bronchoesophagologist, thus providing an opportunity for the establishment of a diagnosis by the pathologist. The estimated frequency of fungus infections as found in material in the Registry of Otolaryngic Pathology and as reported in the literature will also be presented, with special reference to cases in which the diagnosis was made from lesions presenting in these areas.

EXHIBIT S-15
CHONDRODERMATITIS NODULARIS CHRONICA HELICIS

Exhibitor:
Elson B. Helwig, M.D.
BY INVITATION
Armed Forces Institute of Pathology, Washington, D.C.

Co-Exhibitor:
Richard Shuman, M.D.
BY INVITATION

The exhibit will show colored transparencies of the pathologic changes occurring in chondrodermatitis nodularis chronica helicis. The exhibit is based upon the clinical pathologic correlation of one hundred lesions.

EXHIBIT S-16
ORGANISMS IDENTIFIED IN INFLAMMATORY LESIONS OF THE EYES IN THE REGISTRY OF OPHTHALMIC PATHOLOGY, ARMED FORCES INSTITUTE OF PATHOLOGY, WASHINGTON, D.C.

Exhibitor:
Helenor Campbell Wilder
Armed Forces Institute of Pathology, Washington, D.C.

Co-Exhibitor:
Major John H. Bickerton, (MC) USAF
BY INVITATION

The exhibit will consist of color transparencies of some of the organisms, including worms, protozoa, fungi and bacteria, found in ocular lesions and of the inflammatory processes which they incite, with a brief statistical survey of the cases in the Registry of Ophthalmic Pathology in which such organisms have been identified.

EXHIBIT S-17
VERTICAL OCULOMOTOR PROBLEMS

Exhibitor:
Walter H. Fink, M.D.
Minneapolis, Minn.

The exhibit will be a summary of our present-day knowledge pertaining to this important, and as yet controversial, field The presentation will consist of two parts: (1) Basic data—embryology, comparative anatomy, anomalies, gross anatomy, physiology and pathology. (2) Clinical data—frequency, diagnosis, management and final results. Charts, diagrams, photographs and specimens will be used in the presentation.

EXHIBIT S-18
SELECTED PROBLEMS IN THE DIAGNOSIS OF LARYNGEAL CANCER

Exhibitor:
Stanton A. Friedberg, M.D.
Department of Otolaryngology, University of Illinois College of Medicine, Chicago; Presbyterian Hospital, Chicago; and Veterans Administration Hospital, Hines, Ill.

Co-Exhibitor:
Linden J. Wallner, M.D.

The exhibit consists of approximately 60 enlarged kodachrome views of the larynx with illustrations of the following problems: (1) leukoplakia and cordal carcinoma; (2) the relation between appearance and actual extent of carcinoma (a comparison of views of the lesion with the operative specimen); (3) the lack of symptoms in extracordal carcinoma; (4) variations in diagnostic aids; (5) problems in the choice of therapy; (6) carcinoma of the larynx and tuberculosis.

EXHIBIT S-19
DISEASES OF THE EAR DRUM. A NEW TECHNIC OF TEACHING DISEASES OF THE MIDDLE EAR

Exhibitor:
Irvin Hantman, M.D.
School of Medicine, Georgetown University, Washington, D.C. and Episcopal Ear, Eye and Throat Hospital, Washington, D.C.

This exhibit consists of natural colored photographs of the tympanic membrane, as viewed through flesh-colored moulages of the auricle and the external ear canal. Special ear specula, into which have been mounted magnifying lenses, are used to view the tympanic membranes. The entire effect is that of examining the eardrum head through a speculum in the external canal as in real life. Each tympanic membrane is accompanied by an artist's drawing in color of that condition, with each point of interest carefully explained. Various groups of diseases are shown, including normal drumheads, acute and chronic inflammations, traumatic ruptures, secretory otitis media with sequences depicting movement of the fluid level under various conditions and after aspiration. This exhibit should be of considerable interest to those who are interested in teaching medical students, interns and residents and to general practitioners.

EXHIBIT S-20
CLINICAL AUDIOLOGY AND HEARING REHABILITATION
Exhibitor:
Marion P. Downs
BY INVITATION
University of Denver, Denver, Colo.

A filmstrip, "Out from Silence," consisting of colored cartoons to illustrate the nature of hearing and hearing loss, the prevention of hearing loss, and the rehabilitation of the hard of hearing, will be shown. Posters, photographs and charts will illustrate present practice and recent advances in clinical audiology.

EXHIBIT S-21
MAXILLOFACIAL PLASTIC SURGERY
Exhibitor:
Jacques W. Maliniac, M.D.
New York Polyclinic Medical School and Hospital, New York, N.Y.

The exhibit consists of charts and transparencies illustrating plastic repair of maxillofacial deformities of congenital and acquired origin. Stress is placed upon the prevention and early repair of childhood deformities. Radiation sequelae of benign and malignant lesions of face and advantages of surgical repair over radiation therapy in certain conditions will be demonstrated.

EXHIBIT S-22
RADICAL NECK DISSECTION IN OTOLARYNGOLOGY
Exhibitor:
Samuel Kaplan, M.D.
Veterans Administration Hospital
Long Beach, Calif.

Drawings of stages in radical neck dissection for carcinoma of the laryngopharynx, mouth, tongue, and tonsils will be shown. Mounted excised specimens of en block surgical excisions and colored transparencies of lesions treated by this method, will be presented.

EXHIBIT S-23
HISTORICAL LANDMARKS IN OPHTHALMOLOGY
Exhibitor:
Elliott Baldwin Hague, M.D.
Millard Fillmore Hospital, Buffalo, N.Y.

This exhibit comprises books and related items selected from a private collection of ophthalmologic classics, chosen from the standpoint of their relative importance and their suitability as exhibit material. Included will be most of the ophthalmologic subjects from the Garrison-Morton list, as well as even more rare material, more or less unique, such as a 13th century Albucasis manuscript containing a number of references to ophthalmic surgery, with illustrations of instruments.

EXHIBIT S-24
TUMORS OF THE LACRIMAL GLAND
Exhibitor:
Arnold W. Forrest, M.D.
BY INVITATION
Institute of Ophthalmolgy of the Presbyterian Hospital, and Columbia University College of Physicians and Surgeons, New York N.Y.
Co-Exhibitors:
Ira S. Jones, M.D.
Raymond Pfeiffer, M.D.

This exhibit will include photographs, roentgenograms, and photomicrographs illustrating the salient features of various types of lacrimal gland tumors.

EXHIBIT S-25
PRACTICAL GRADUATE INSTRUCTION IN HISTOPATHOLOGY OF THE EYE
Exhibitor:
Brittain F. Payne, M.D.
New York University Post Graduate School, and New York Eye and Ear Infirmary, New York, N.Y.
Co-Exhibitors:
A. Marvin Gillman, M.D.
Gerald B. Kara, M.D.
Bernard A. Roberts, M.D.

An exhibit of finished drawings of normal and pathologic sections of eyes by graduate students in ophthalmology will be presented. Other illustrations will show student progress in the course. Actual specimens will be used to demonstrate various diseases. The constant use of the ATLAS AND TEXTBOOK OF OPHTHALMIC PATHOLOGY will be emphasized by display.

Exhibit S-26
NEW METHODS AND NEW INSTRUMENTS
Exhibitor:
American Academy of Ophthalmology and Otolaryngology

New methods and instruments presented at any of the Scientific Sessions on Ophthalmology or Otolaryngology can be placed on display here.

TECHNICAL EXHIBITORS — 1953

Exhibitor	Booth No.
Alcon Laboratories, Inc.	37
The Alkalol Co.	29
Allied Instrument Manufacturers, Ltd.	R-14
American Cystoscope Makers, Inc.	87 & 88a
American Optical Co.	7 thru 14
Audiometer Sales Corp.	47
Audivox, Inc.	42
Aurex Corp.	R-3
Barnett Optical Laboratories	37
Bausch & Lomb Optical Co.	93 thru 100
Bay State Optical Co.	106
Austin Belgard, Inc.	18 and ½ of 19
Bell & Howell Co.	103 & 105
N. P. Benson Optical Co.	84 & 85
The Blakiston Co.	83
Boll & Lewis Optical Co.	41
Burroughs Wellcome & Co., Inc.	117
George H. Butterfield & Son	R-9
Camel Cigarettes	27 & 28
Cameron Surgical Specialty Co.	74
Carroll Dunham Smith Pharmacal Co.	R-8
Ciba Pharmaceutical Products, Inc.	77
ClerSite Co.	"C"
The Coca-Cola Co.	26
Coreco Research Corp.	59
Davis & Geck, Inc.	R-6
The DeVilbiss Co.	15 & 16
Doho Chemical Corp.	34
Eaton Laboratories, Inc.	R-15
Encyclopaedia Britannica	R-7
Encyclopedia Americana	72
Grieshaber Swiss Eye Instruments	38
Grune & Stratton, Inc.	35
Guild of Prescription Opticians of America, Inc.	43 & 44
Paul B. Hoeber, Inc.	32
Hollister-Stier Laboratories, Inc.	46
The House of Vision, Inc.	60, 61 & 62
Hudson Optical Corp.	104
Ion Optical Co.	R-5
Jenkel-Davidson Optical Co.	108
Keeler Optical Products, Ltd.	90 & 91a
Keystone View Co.	23 & 24
Kono Manufacturing Co.	76
Lea & Febiger	51
Liberty Optical Manufacturing Co.	R-17
J. B. Lippincott Co.	102
P. Lorillard Co.	36A
Mager & Gougelman, Inc.	20 and ½ of 19
The Maico Co., Inc.	66 & 67
Marcelle Cosmetics, Inc.	54
Medical Case History Bureau	"D"
Medical Film Guild, Ltd.	I. I.
Merck & Co., Inc.	114
E. B. Meyrowitz Surgical Instruments Co., Inc.	79 & 80
Miller Surgical Co.	91b
Modern Optics, Inc.	115 & 116
C. V. Mosby Co.	86
V. Mueller & Co.	1 thru 5

Exhibitor	Booth No.
Nepera Chemical Co., Inc.	58
New Era Optical Co.	R-19 & R-20
New Jersey Optical Co.	48
Novocol Chemical Manufacturing Co., Inc.	68
Ohrig Laboratories, Inc.	109
Ophthalmos, Inc.	R-1
Paravox, Inc.	R-13
Chas. Pfizer & Co., Inc.	9
Philip Morris & Co. Ltd., Inc.	23
George P. Pilling & Son Co.	63, 64 & 65
Precision-Cosmet Co.	"B"
The Professional Press, Inc.	50
Radium Chemical Co., Inc.	30
Ritter Co., Inc.	69 thru 73
W. B. Saunders Co.	19
Schering Corp.	33
Schieffelin & Co.	R-11
Shuron Optical Co., Inc.	81 & 82
Smith, Kline & French Laboratories	45
Soft-Lite Lens Co., Inc.	107
Sonotone Corp.	78
Storz Instrument Co.	110, 111 & 112
Sturgis-Grant Productions, Inc.	R-2
Surgical Mechanical Research	88b & 89
The Thermoscetor Co.	31
Titmus Optical Co., Inc.	35
Travenol Laboratories, Inc. (Subsidiary of Baxter Laboratories, Inc.)	R-21 & R-22
Uhlemann Optical Co. of Illinois	52 & 53
The Univis Lens Co.	39 & 40
U. S. Recording Co.	R-4
Victory Optical Manufacturing Co.	101
The Visionez Corp.	R-12
Dr. Max M. von Stirne	R-18
Ward Manufacturing Co.	R-10
Welch Allyn, Inc.	17
White-Haines Optical Co.	21 & 22
The Williams & Wilkins Co.	36
Winthrop-Stearns, Inc.	25
Carl Zeiss, Inc.	R-16
Zenith Radio Corp.	55
Zylite Products Co., Inc.	113
Zylo Ware Corp.	118

TECHNICAL EXHIBITS

The Academy is appreciative of the cooperation of all technical exhibitors.

Booths with numbers preceded by "R" are located in the Red Lacquer Room.

Alcon Laboratories, Inc., Fort Worth, Texas **Booth 57**

Physicians are cordially invited to visit Alcon's exhibit to examine and discuss sterile ophthalmic solutions of their manufacture. Sterile Methyl Cellulose solutions for ophthalmic use alone, and in combination with other ophthalmic drugs will be featured. Other ophthalmic solutions, namely, Op-thal-zin and Zincfrin will be shown, along with Alcon's physiologic and nonirritating nasal decongestant, Alcon-efrin.

The Alkalol Company, Taunton, Mass. **Booth 29**

The Alkalol Company is featuring ALKALOL, a solution especially designed for the treatment of the mucous membranes of the eye, nose and throat.

First compounded in 1896, its balanced formula of salts and essential oils makes a solution that is effective and at the same time nontoxic, mild and pleasant. It is alkaline and a mucus solvent.

ALKALOL is an excellent vehicle for many drugs. For further information, please call at Booth 29.

Allied Instrument Manufacturers, Ltd., London, England **Booth R-14**

In addition to a comprehensive range of ophthalmic and orthoptic instruments including the Projection Perimeter and Keith Lyle's Major Amblyoscope, doctors are invited to examine and criticize the following new instruments. A modification of Professor Amsler's Keratograph, as made for the Institute of Ophthalmology; a co-axial Wide Angle Hruby lens, for stereoscopic biomicroscopy of the fundus; the Aimeye, a teaching aid for slit lamp microscopy and ophthalmoscopy.

American Cystoscope Makers, Inc., New York, N. Y. **Booths 87 & 88a**

Precision instruments for diagnosis and operative procedures will be on display in our booth. These include nasopharyngoscopes of advanced design, thoracic electroresection equipment, low-reflection treated telescopes for bronchoscopy and esophagoscopy, together with equipment for endoscopic photography. Representatives will be in attendance to demonstrate these and to answer any questions which may arise. A cordial invitation is extended to members and guests of the Academy.

American Optical Co., Southbridge, Mass. **Booths 7 thru 14**

The American Optical Company, Instrument Division, will have a display of the latest models of ophthalmic instruments and equipment, including a new attachable Phoroptor Illuminator, a new test lens cabinet with automatic illumination, the Schepens Binocular Indirect Ophthalmoscope, the latest line of ophthalmic diagnostic instruments, as well as other new items used in examination and refraction of the eyes.

It will have some late developments in scientific instruments, such as microscopes, and will also have on display a full line of various types of bifocal and trifocal lenses, plastic implants, and eye, ear, nose, and throat surgical instruments.

There will be trained instrument representatives on hand who will cheerfully explain and demonstrate any of these instruments or ophthalmic equipment. Members and guests of the Academy are cordially invited to visit these booths.

Audiometer Sales Corp., Minneapolis, Minn. **Booth 47**

The Audiometer Sales Corporation welcomes your visit to its exhibit of ADC

Audiometers. Featured will be the Model P60A speech audiometer. Designed for your convenience, this diagnostic instrument has a built-in speaker, monitor receiver and talkback circuit, making it equally suitable for office or sound room use. Precision and accuracy, as in all other ADC Audiometers, are of the highest quality.

Models 53A, 53C and the well-known 50E2 will also be displayed. Your inspection is invited and a complete demonstration of any model is available at your request.

Audivox, Inc., Boston, Mass. Booth 42

Aurex Corp., Chicago, Ill. Booth R-3

Aurex Corporation is exhibiting hearing aid testing equipment. They are also featuring their new transistor hearing aids.

Barnett Optical Laboratories, Chicago, Ill. Booth 37

Bausch & Lomb Optical Co., Rochester, N. Y. Booths 93 thru 100

Bay State Optical Co., Westbury, L.I., N. Y. Booth 106

Austin Belgard, Inc., Chicago, Ill. Booth 18 & ½ of 19

Bell & Howell Co., Chicago, Ill. Booths 103 & 105

N. P. Benson Optical Co., Minneapolis, Minn. Booths 84 & 85

The Blakiston Co., New York, N. Y. Booth 83

Available for examination at Blakiston's booth will be page proofs on the forthcoming **New Edition** of **Wolff's** "Anatomy of the Eye." Other distinguished Blakiston volumes on the eye, including **Meller's New Sixth Edition** "Ophthalmic Surgery," will be shown. Also on display will be two new anatomies which offer special sections on the eye: **Krieg's New Second Edition** "Functional Neuroanatomy" and the **New Eleventh Edition** of "Morris' Human Anatomy," edited by J. Parsons Schaeffer, M.D.

Boll & Lewis Optical Co., Chicago, Ill. Booth 41

Burroughs Wellcome & Co., Inc., Tuckahoe, N. Y. Booth 117

Burroughs Wellcome & Co., Inc., will feature AEROSPORIN® OTIC SOLUTION STERILE which is hygroscopic, has an acid pH, and is antimicrobial, especially against Pseudomonas aeruginosa. It contains AEROSPORIN® SULFATE POLYMYXIN B SULFATE with Propylene Glycol Otic Solution Sterile.

AERODRIN brand INTRANASAL SOLUTION, which produces local decongestion without central stimulation and provides broad-spectrum antisepsis, will also be shown.

George H. Butterfield & Son, Portland, Ore. Booth R-9

George H. Butterfield & Son will exhibit the Para Curve Corneal Contact Lenses and the Para Scleral Fluidless Corneal-Scleral Contact Lenses.

Camel Cigarettes, New York, N. Y. Booths 27 & 28

CAMEL Cigarettes will mark your initials on an attractive plastic cigarette case filled with a package of those mild, flavorful CAMELS. This exhibit features a display of some of the tobaccos used in blending this famous cigarette which leads all other brands by many billions.

Cameron Surgical Specialty Co., Chicago, Ill. Booth 74

See the Anthony-Fisher Suction Coagulating Handle and Electrodes for nasal hemorrhage; cauteradios and cauterodynes for ocular, aural, nasal and oral electrosurgery, electrocauterization and electrocoagulation; the Boros Flexible Esophagoscope and Flexible Gastroscopes; electrodiagnostic lamps and instrument sets; mirror-type headlights; binocular spectacle loupes and other specialties for ophthalmology and otolaryngology.

Carroll Dunham Smith Pharmacal Co., New Brunswick, N. J. Booth R-8

We will feature Lipotriad, a product with unusually potent lipotropic oxytropic principles. Our representative will welcome the opportunity of discussing the value of Lipotriad in senile macular degeneration, diabetic retinopathy and other ophthalmic conditions.

Ciba Pharmaceutical Products, Inc., Summit, N. J. **Booth 77**

ClerSite Co., Chicago, Ill. **Booth "C"**

The Coca-Cola Co., Atlanta, Ga. **Booth 26**

Ice-Cold Coca-Cola served through the courtesy and cooperation of the Coca-Cola Bottling Company of Chicago, Inc., and the Coca-Cola Company.

Coreco Research Corp., New York, N. Y. **Booth 59**

Davis & Geck, Inc., Brooklyn, N. Y. **Booth R-6**

Davis & Geck, Inc., manufacturers of sutures and other surgical specialties, will exhibit the most comprehensive line of eye sutures available anywhere. These products have been developed in collaboration with outstanding ophthalmologists throughout the world. Both sutures and needles have been designed and perfected to meet the exacting conditions encountered in eye surgery. Exceptional sharpness of needle points will be demonstrated. Of special interest will be Davis & Geck's new Aureomycin Dressing and Aureomycin Packing for various surgical procedures.

The DeVilbiss Co., Somerset, Pa. **Booths 15 & 16**

The Devilbiss Company welcomes you to its exhibit of professional and prescription types of atomizers, nebulizers and vaporizers. Three new additions to our line, the new no. 146 all-metal Vaporizer, the no. 840 Continuous Flow Nebulizer for sustained aerosol treatment, and the no. 41 Pocket Neublizer with carrying case, will be available for evaluation.

Doho Chemical Corp., New York, N.Y. **Booth 34**

Doho Chemical Corporation is pleased to exhibit AURALGAN, the ear medication for the relief of pain in Otitis Media and removal of Cerumen; RHINALGAN, the nasal decongestant which is free from systemic or circulatory effect and equally safe to use on infants as well as the aged; and the NEW OTOSMOSAN, the effective, nontoxic ear medication which is Fungicidal and Bactericidal (gram negative-gram positive) in the suppurative and dermatomycotic ears. Mallon Chemical Corporation, subsidiary of the Doho Chemical Corporation, is also featuring RECTALGAN, the liquid topical anesthesia, also bactericidal and fungicidal for control of secondary invaders, particularly recommended for treatment of mold infections (Monilia) occurring after antibiotic therapy; also for relief of pain and discomfort in hemorrhoids, pruritus and perineal suturing.

Eaton Laboratories, Inc., Norwich, N. Y. **Booth R-15**

For the treatment of ophthalmic infections, the recently Council-accepted preparations will be on display: Furacin Ophthalmic Liquid and Furacin Ophthalmic Ointment. These preparations are effective antibacterial agents and do not irritate the eye or delay healing.

Other E.E.N.T. dosage forms of Furacin® are Furacin Nasal plain, Furacin Nasal with ephedrine, Furacin Nasal with Neo-Synephrine® and Furacin Anhydrous Ear Solution.

The most recent preparation is Furacin Soluble Powder, a convenient preparation for treating external otitis.

Encyclopaedia Britannica, Chicago, Ill. **Booth R-7**

Encyclopedia Americana, Chicago, Ill. **Booth 92**

Grieshaber Swiss Eye Instruments, Chicago, Ill., **Booth 38**

Exclusive authorized distributor for Ernst Grieshaber, Switzerland.

If you are not familiar with the genuine Grieshaber Eye Knives, which like the famous Grieshaber Corneal Needles cut with amazing accuracy and lightness of touch, we urge you to try them. These knives have been perfected through years of conscientious scientific research on the part of Dr. Hans Grieshaber, Shaffhausen, Switzerland, in collaboration with the world's leading ophthalmic surgeons.

Samples of various popular Swiss-made Eye Knives, Cataract Knives, Trephines

and Keratomes will be displayed, as well as the original and inimitable cutting edge Corneal Needles; also the Arruga Stainless Steel Needle Holder used in conjunction with the Corneal Needles.

Attention is directed to the fact that these instruments are not mass produced, and in order for us to expedite delivery of your orders, it is advisable to order promptly.

Grune & Stratton, Inc., New York, N. Y.
Booth 35

Mr. Frank Kurzer will again be pleased to see you and show you the newest titles, among which are Tower's **Differential Diagnosis of Common Diseases of the Eyeground,** Myklebust's **Auditory Disorders in Children,** Spiegel's **Progress in Neurology and Psychiatry, Vol. VIII,** and Rubinstein's **The Study of the Brain.** Of particular interest is the division, in its second printing, of **Progress in Ophthalmology and Otolaryngology** (edited by Wiener, Ireland, et al.) into two books —one for each specialty. Fox's **Ophthalmic Plastic Surgery,** Linksz's **Physiology of the Eye,** Wiener and Scheie's **Surgery of the Eye** (ed. 3) and many others will also be shown.

Guild of Prescription Opticians of America, Inc., New York, N. Y.
Booths 43 & 44

Paul B. Hoeber, Inc., New York, N. Y.
Booth 32

Hollister-Stier Laboratories, Inc., Wilkinsburg, Pa. **Booth 46**

Trained and experienced technicians will be in attendance at all times. Accepted methods of diagnosis and therapy will be visually presented with emphasis on technic and the mechanics of allergy.

The complete allergy service of Hollister-Stier Laboratories offers antigens prepared from special dust, pollens, foods, environmental inhalants and molds. Various diluents will be explained in detail.

The House of Vision, Inc., Chicago, Ill.
Booths 60, 61 & 62

The House of Vision will again display new and different equipment, in addition to the domestic and foreign instruments which created so much interest at the last Academy meeting.

A new item to be featured is the Universal revolving stage, mounted on a hydraulic table, which enables the ophthalmologist to perform all his examinations without changing his own position or that of the patient. The hydraulic table is divided into four sections, each fitted with sockets, switches and control lamps, to accommodate almost any four instruments desired. A handle under the revolving stage pulls out a fixed working plate to a horizontal position in front of the patient. The table is revolved until the correct instrument is in position before the working plate. Then the instrument is simply rolled out on to the plate.

Another new item to be exhibited is the Haas target wand for field examination, designed for greater accuracy and detection of malingering. It features a retractable target installed in a black wand. The target pin can be made to appear or disappear any time during a field examination.

Haag-Streit, of Switzerland, now has the Goldmann Weekers adaptometer ready for production. This instrument is reported to be the ultimate in dark adaptation testing and recording. It is designed to examine every possible function of the dark-adapted eye under all conditions. Other Haag-Streit instruments will also be on display—among them the Goldmann slit lamp with all attachments, the Goldmann perimeter, the Javal Schiotz ophthalmometer and the Amsler ophthalmoscope.

The Zeiss-Opton slit lamp will be shown; also the Krahn slit lamp, Aimark slit lamp, several different types of lensometers, and the latest diagnostic instruments such as ophthalmoscopes, retinoscopes and hand slit lamps.

A variety of other new and unusual aids to the ophthalmologist will be available for inspection, including skiascopy racks, lamps, children's trial frames and many others.

Lenses designed and ground in very rare and difficult corrections will be displayed, including the "Mi-focal," a new bifocal developed by The House of Vision for strong myopic corrections.

Hudson Optical Corp., New York, N. Y.
Booth 104

Ion Optical Co., Detroit Mich.
Booth R-5

The Ion Optical Company recognizes the importance of the accurately compounded prescription.

We will have on exhibit, lenses designed and fabricated in our modern laboratory for unusual prescriptions. Of special interest to the ophthalmologist will be our illustration of the importance of proper dispensing technics for the patient's comfort, both cosmetically and optically.

In addition, we will present a selection of custom frames in a unique display cabinet, suitable for the professional office.

Jenkel-Davidson Optical Co., San Francisco, Calif. Booth 108

Jenkel-Davidson, dispensing opticians, will exhibit ophthalmic instruments and accessories designed and manufactured for the ophthalmologist exclusively. This exhibit will include the Harrington Black Light Perimeter, the Black Light Tangent Screen Illuminator, a completely new series of motor driven refractor stands and lift tables, the J-D Clinicamera for external eye photography, gonioscopy ceiling fixture, Transport Transformer and several other recently developed ophthalmic devices.

Keeler Optical Products, Ltd., London, England Booths 90 & 91a

The KEELER exhibit aims to include special instruments developed and perfected particularly for the American specialist in ophthalmology and otolaryngology. Demonstrated for the first time in the United States will be the following:

THE HALLPIKE-BLACKMORE MONOCULAR EAR MICROSCOPE from prototypes originated in the Otological Research Unit of the Medical Research Council, this new otoscope provides intense shadowless 12 volt illumination and a special optical system to give dazzle-free vision of the entire tympanic membrane at optimum x6 magnification. It permits critical definition of abnormalities which have hitherto been almost invisible by existing methods of examination.

THE ASHER-LAW STEREOSCOPE incorporates all the features necessary in theory and sufficient in practice for an effective stereoscope. With it the operator can control accommodation only, convergence only, accommodation and convergence in any desired relation, normal and abnormal.

THE KEELER STREAK RETINOSCOPE has now been produced in response to a demand for a Streak Retinoscope of the Keeler standard of performance. Speedy accurate determination of cylinder axis and sharply defined reflex characterize this retinoscope.

THE KEELER HEADBAND TRIAL FRAME: particularly useful for children and high myopes, and for postcataract cases. The three-cell frame is affixed to a lightweight headband by means of a double-ball joint enabling the lenses to be mounted on any desired plane or at any distance from the cornea.

Other instruments of the KEELER range of constant appeal to the specialist include the PANTOSCOPE; KEELER MAGNIFYING SPECTACLE; THE MEASURING OPHTHALMOSCOPE providing for three-dimensional quantitative estimations in millimeters on the fundus, and THE "CONTINENTAL" OPHTHALMOSCOPE designed for direct and indirect ophthalmoscopy.

In addition to the above, KEELER staff will be pleased to demonstrate other standard instruments and equipment of the KEELER range.

Keystone View Co., Meadville, Pa.
Booths 23 & 24

Two new developments in the field of binocular testing materials will be shown at the Keystone View Company's exhibit.

A new binocular instrument, the Orthoscope, embodying lenses adjustable for interpupillary distances will be of special interest. The flat field obtained makes the instrument of special value in screening aniseikonia cases.

The new O'Brien Field Screening Stereograms designed to detect central scotomas and gross field defects will be exhibited and demonstrated.

In addition to these new items Keystone Home Training Services, the Dvorine Animated Fusion Charts and other orthoptic training materials and equipment will be on display.

Kono Manufacturing Co., Woodside, N. Y.
Booth 76

In keeping with their new emphasis on cosmetic aids in optical frames. The Kono Manufacturing Company's exhibit is one of elegance. Against a setting of coral tufted walls and modern furnishings, highlighted with touches of turquoise and sea green, Kono's complete line of women's frames, men's frames, and children's frames will be shown.

Of particular interest will be the newest Konoptics designed with Kono's new EMPAHTONE® bridge. This clear or phantom bridge has been incorporated in Kono's frame as an aid to attenuate a wide nose and lengthen a short nose. Another cosmetic aid which will be featured is the artful blending of phantom and solid zyl in a two-tone combination. This technic is designed to decrease and de-emphasize the dark circles and tiny wrinkles under and around the eyes.

For added convenience, all of Kono's frames will be easily and conveniently seen in a unique revolving showcase which is finished in a natural wood to blend with the surrounding fixtures.

One section of the exhibit is devoted to a fitting salon where visitors may relax in a luxurious modern setting and examine Kono frames first hand.

Lea & Febiger, Philadelphia, Pa.
Booth 51

Liberty Optical Manufacturing Co., Newark, N. J. **Booth R-17**

Joseph G. Bianco, General Manager of Liberty Optical Company, reports that their exhibit will cofeature the new Pencil Stripe and 3-D frames. Pencil Stripe is an entirely new fashion concept in frame creation, available in both Ladies' and Men's sizes. 3-D is a novel and revolutionary frame whose design provides for recessed lenses giving it a new, functional beauty plus protection against scratching. 3-D is being made for men only at this time. We recommend that you devote a few minutes of your time at the convention to see these and other Liberty exclusives in Booth R-17.

J. B. Lippincott Co., Philadelphia, Pa.
Booth 102

P. Lorillard Co., New York, N. Y.
Booth 50A

P. Lorillard Company, manufacturers of OLD GOLD and EMBASSY Cigarettes as well as BRIGGS Pipe Mixture and other famous tobacco products, will exhibit and demonstrate their new KENT Cigarette with the exclusive Micronite Filter, which takes out up to seven times more nicotine and tars than other filter cigarettes.

Mager & Gougelman, Inc., Chicago, Ill.
Booth 20 & ½ of 19

Mager & Gougelman, Inc.'s technical exhibit will consist of the finest assortment of artificial eyes in both glass and plastic. The display will also include all types of motility implants, sphere implants, radiation therapy eye shields, intraocular foreign body locators, drains and conformers. In addition, the exhibit will feature facial restorations and orbital appliances.

The Maico Co., Minneapolis, Minn.
Booths 66 & 67

For 1953, Maico will present its expanded line of medical acoustic instruments including clinical, professional and industrial audiometers, hearing aids and special instruments for auditory and speech training.

We will feature: TRANSIST-EAR—the revolutionary, all-transistor hearing instrument using a **dime-size** energy capsule in place of the larger batteries for power. SPEECH PEDESTAL AUDIOMETER—for use of recorded speech material. PSYCHOMETER—which uses psychogalvanic reaction to determine hearing threshold of very young children, malingerers and those suffering from psychogenic deafness. CHROMALIZER—helps teach deaf children by substituting a visual spectrum for sound spectrum.

Marcelle Cosmetics, Inc., Chicago, Ill.
Booth 54

New developments and new products in the Marcelle hypo-allergenic Cosmetic line will be demonstrated at the Marcelle exhibit. Representatives will include members of the Marcelle research organi-

zation who will give data and information on the role of Marcelle hypo-allergenic Cosmetics in the physician's practice.

Medical Case History Bureau, New York, N. Y. Booth "D"

A handy, compact method for keeping patients' complete eye, ear, nose and throat case histories is featured here. In the INFO-DEX RECORD SYSTEM, charts are so arranged on folders that subsequent cards may be attached by means of a cotter pin which holds the cards together in correct sequence. Different colored cards for various types of data, such as refraction, audiogram, allergy, laboratory, x-ray findings, are obtainable. Included in this system is an automatic diagnostic cross-indexing. Steel filing cabinets in which to house these history charts and bookkeeping records are also displayed. Representatives at the exhibit will gladly consult with you on any record problem or help you systematize your office routine.

Medical Film Guild, Ltd., New York, N. Y. Booth L-L

Merck & Co., Inc., Rahway, N. J. Booth 114

Merck & Co., Inc. is featuring the ophthalmic forms of CORTONE (Ophthalmic Ointment 1.5%; Ophthalmic Suspensions 0.5% and 2.5%; and Ophthalmic Ointment of CORTONE with Bacitracin); FLOROPRYL (0.1%—Peanut Oil—5 cc. dropper bottle); CARCHOLIN (0.5% gm. bottle) and other ophthalmic preparations.

E. B. Meyrowitz Surgical Instruments Co., Inc., New York, N. Y. Booths 79 & 80

E. B. Meyrowitz Surgical Instruments Co., Inc., will have on display a complete line of eye, ear, nose and throat instruments of our own and foreign manufacture for both surgery and diagnosis. Included will be all the newer surgical items as well as the Chamlin Perimeter, which has not been publicly shown before.

Miller Surgical Co., Chicago, Ill. Booth 91b

Miller Surgical Company will show leading illuminated diagnostic equipment: Otoscopes, Ophthalmoscopes, Eyespuds with Magnet, Transillumination Lamps, Headlights. Also, the Miller Electro-scalpel Model 10V-O, a complete office unit which cuts, desiccates, fulgurates, coagulates, for most delicate work up to light major surgery. Portable, weight 19 lbs. with cords, handles, and foot switch. Also accessories such as Insulated Snares, Smoke Ejector Suction Fulgurator.

Modern Optics, Inc., New York, N. Y. Booths 115 & 116

Modern Optics, Inc., will have one of their usual interesting exhibits.

How high-fidelity definition, through segments free from aberration or distortion, is produced by MODERN'S exclusive Threshold-Welding process will be an important feature...together with the popular MODERN Lenticular Cataract Lens Series, and the many other interesting MODERN lens specialties.

C. V. Mosby Co., St. Louis, Mo. Booth 86

V. Mueller & Co., Chicago, Ill., Booths 1 thru 5

V. Mueller & Company will present a large and unusually interesting exhibit of fine instruments and equipment. It will include complete lines of instruments for eye surgery, fenestration and other ear surgery, rhinoplasty, adenoidectomy and soft palate work, laryngeal surgery and bronchoscopy. Selected samples of the most modern office and surgical equipment will also be shown, including the all-new Herb-Mueller Explosion-Proof Ether-Vacuum Unit with its new automatic recirculating lubrication system, and other Mueller pumps.

Nepera Chemical Co., Inc., Yonkers, N. Y. Booth 58

New Era Optical Co., Chicago, Ill. Booths R-19 and R-20

New Jersey Optical Co., Irvington, N. J. Booth 48

Novocol Chemical Manufacturing Co., Inc., Brooklyn, N. Y. Booth 68

A cordial invitation is extended to all to visit with us. A qualified representative will be in attendance to explain the many advantages to be enjoyed with the use of MONOCAINE HYDROCHLORIDE solution (for local anesthesia) and the ANESTUBE SYSTEM. The growing popularity of MONOCAINE HYDROCHLORIDE solution and the ANESTUBE SYSTEM with ophthalmologists, otolaryngologists and plastic surgeons merits a few minutes of your time at booth 68.

Obrig Laboratories, Inc., New York, N. Y. Booth 109

In addition to the exhibit of solutionless contact lenses, The Obrig Laboratories, Inc., will issue a paper on the success of contact lenses in unilateral and bilateral aphakia.

Ophthalmos, Inc., Union City, N. J. Booth R-1

Ophthalmos, Inc. cordially invites you to visit their booth displaying latest developments in ophthalmic solutions by Robert R. Feinstein Research Associates. We are featuring laboratory controlled prepackaged ophthalmic prescriptions to be dispensed in their original dropper sealed containers, assuring sterility, accuracy and maximum comfort.

METH I SOL—A replacement for tears in lacrimal deficiencies. It has the same index of refraction as the cornea and also approximates the viscosity of tears.

PILOCARPINE Solutions—1% and 2%.

PROP I SOL—Sodium Propionate 10% solution. A wide spectrum physiologic antibiotic.

VAS I ZINC—Zinc Sulfate ¼% solution in high viscosity buffered vehicle.

ZINC I ZOL—Zinc Sulfate ¼% and Phenylephrine HCL ⅛% in high viscosity buffered solution.

Also featured will be improved and modernized solutions of drugs which are used by you daily in your examining room.

FLUORESCEIN SODIUM 2%—Stabilized and preserved against contamination.

TETRACAINE HCL ½%—The popular topical anesthetic in a superior form.

MYDR I ATIN—Phenylephrine HCL 5% and Eucatropine HCL 5% in an improved vehicle. Nonirritating, rapid acting, stable mydriatic.

Mr. Robert R. Feinstein will be available to discuss cooperating in research or clinical projects you may contemplate.

Paravox, Inc., Cleveland, Ohio Booth R-18

The Paravox, Inc., exhibit will display Paravox Hearing Aids, including the "TRANSonic," the All-Transistor Hearing Aid. Great interest has been shown in this new type of instrument which operates on one battery and does not employ vacuum tubes. Photoscription, the fitting of the aid through use of photograph of the aid's soundwaves, will also be featured.

Chas. Pfizer & Co., Inc., Brooklyn, N. Y. Booth 6

Philip Morris & Co. Ltd., Inc., New York, N. Y. Booth 75

Philip Morris and Company will show the results of research on the irritant effects of cigarette smoke. These results show conclusively that Philip Morris cigarettes are less irritating than other cigarettes. An interesting demonstration will be made on smokers at the exhibit which will show the difference in cigarettes.

George P. Pilling & Son Co., Philadelphia, Pa. Booths 63, 64, & 65

Pilling of Philadelphia will again show a complete line of instruments which will interest the ophthalmologist as well as the otolaryngologist. Of special note are the Broyles Suction Nasal and Laryngeal Forceps, Schwartz Multi-purpose Laryngeal Forceps in various angles and sizes, and Jackson Bronchoscopic Telescopes and Magniscopes.

Precision-Cosmet Co., Minneapolis, Minn. Booth "B"

All products manufactured and distributed by Precision will be on display. Particular emphasis will be placed on Precision Certified Multifocal lenses, an entirely new type of bifocal and trifocal, that will be shown at the Academy for the first time. Glass-Gard and Mann Deodorants will be demostrated.

The Professional Press, Inc., Chicago, Ill.
Booth 50

The Professional Press will present "The Eye, Ear, Nose & Throat Monthly," the practical journal for the eye, ear, nose and throat specialist. This journal contains, besides original contributions, a number of feature departments conducted by an outstanding editorial group. Read The Monthly and keep abreast of the newer developments in the eye, ear, nose and throat field.

Radium Chemical Co., Inc., New York, N. Y. **Booth 30**

Radium Chemical Company, Inc., will display the Monel metal radium nasopharyngeal applicator, used for the treatment of hyperplastic lymphoid tissue at the orifice of the eustachian tube and the fossa of Rosenmueller. A complete line of instruments for the application of radon will also be shown.

Ritter Co., Inc., Rochester, N. Y.
Booths 69 thru 73

Displaying motor hydraulically elevated chairs for refraction, examination and treatment.

Two types of completely equipped treatment units, including illuminated instruments, special vacuum and syringe bottles and surgical cuspidor capable of high vacuum volume production.

Specialized office x-ray equipment for accurate radiographs of the paranasal sinuses and allied areas.

A motor hydraulically elevated examination and treatment office chair-table, also a special explosion proof operating room table for eye and plastic surgery.

A four-speed bone surgery engine for fenestration.

Completely automatic office sterilizers, air compressors, and stools complete the exhibit.

W. B. Saunders Co., Philadelphia, Pa.
Booth 49

Once again W. B. Saunders will be proud to be on hand displaying books of interest to ophthalmologists and otolaryngologists.

Please stop by.

Schering Corp., Bloomfield, N. J.
Booth 33

Members of the American Academy of Ophthalmology and Otolaryngology and their guests are cordially invited to visit the Schering exhibits where new therapeutic developments will be featured.

Schering representatives will be present to welcome you and to discuss with you these products of their manufacture.

Schieffelin & Co., New York, N.Y.
Booth R-11

Schieffelin & Co. will introduce its new cycloplegic and mydriatic compound, CYCLOGYL. This product has the ability to produce maximal cycloplegia more rapidly than commonly used preparations. It has the added advantage of allowing return to normal vision within six hours following therapy. CYCLOGYL works equally well in all age groups, including children, and is particularly effective in darkly pigmented irises which usually do not respond to other cycloplegics.

Samples and literature on this product will be distributed to all members of the American Academy of Ophthalmology and Otolaryngology.

Shuron Optical Co., Inc., Geneva, N. Y.
Booths 81 & 82

Smith, Kline & French Laboratories, Philadelphia, Pa. **Booth 45**

The Smith-Kline-French booth will feature SPANSULES—the revolutionary new oral dosage form. Just one SPANSULE, taken on arising, provides a uniform supply of medication throughout the day. Thus, SPANSULES offer you three advantages: (1) smooth uniform action, (2) prolonged therapeutic effect and (3) convenient once-a-day dosage.

Soft-Lite Lens Co., Inc., New York, N. Y.
Booth 107

Sonotone Corp., Elmsford, N. Y.
Booth 78

Sonotone exhibits two new audiometers, one for general diagnostic, the other for advanced diagnostic and research applications. This new line for the

first time offers full facilities to the American otologist for the Luscher Monaural Recruitment and the Denes and Naunton Difference Limen tests now coming into use on the Continent, plus the wide variety of other audiometric tests which are standard in American diagnostic procedures. Also included for the first time are facilities for a continuous check on calibration accuracy as to both frequency and output. Among other features are continuously variable frequency and attenuation, automatic pulsing or interruption, tone amplitude "wobbulation," a variety of masking tones including "white noise," calibrated control of masking-tone intensity, input connections for recorded and "live" speech with a panel meter for monitoring.

Storz Instrument Co., St. Louis, Mo. Booths 110, 111 & 112

Storz Instrument Company will display a complete selection of eye, ear, nose, throat and rhinoplasty surgical instruments. Included will be many new items.

Sturgis-Grant Productions, Inc., New York, N. Y. Booth R-2

Specialized production of scientific teaching films in the fields of ophthalmology and otolaryngology is being demonstrated by the producers of the Academy film, THE EMBRYOLOGY OF THE EYE, and of its companion film, THE EMBRYOLOGY OF THE EAR. The technics employed in producing both "live-action" and "animation" films are graphically illustrated by exhibit material which also indicates the wide variety of projects to which these technics may be applied. Representatives at the exhibit invite discussion of film ideas and production problems.

Surgical Mechanical Research, Los Angeles, Calif. Booths 88b & 89

The Thermosector Co., Oak Park, Ill. Booth 31

The Thermosector, an electrosurgical unit expressly designed for eye, ear, nose and throat technics in office and hospital, will be exhibited and demonstrated. You are invited to investigate the superior features of this device, which is preferred by leading hospitals, clinics and professional men.

Titmus Optical Co., Inc., Petersburg, Va. Booth 56

Travenol Laboratories, Inc. (Subsidiary of Baxter Laboratories, Inc.), Morton Grove, Ill. Booths R-21 & R-22

Travenol Laboratories, Inc., will welcome you at the Piromen exhibit. Extensive laboratory and clinical studies have demonstrated the efficacy, reliability, and safety of Piromen, a new therapeutic agent.

Piromen is a sterile pseudomonas polysaccharide in a colloidal dispersion for parenteral use. Piromen is a stimulant for the endocrine and reticulo-endothelial systems. It has proved its value in the treatment of certain skin disorders, eye disorders, allergies, and angioedema and urticaria associated with penicillin reactions.

Uhlemann Optical Co. of Illinois, Chicago, Ill. Booths 52 & 53

We invite you to see our outstanding display of Glasses by Uhlemann . . . a collection of styles with technical qualities. Our exhibit will include several new features in ophthalmic equipment.

The Univis Lens Co., Dayton, Ohio Booths 39 & 40

United States Recording Co., Washington, D. C. Booth R-4

Victory Optical Manufacturing Co., Newark, N. J. Booth 101

The Visionez Corp., St. Cloud, Minn. Booth R-12

Dr. Max M. Von Stirne, Philadelphia, Pa. Booth R-18

Ward Manufacturing Co., North Arlington, N.J. Booth R-10

We plan to exhibit all of our current popular styles of Zyl Optical Frames for men, women and children, including the first showing of a new style for men in different colors.

Welch Allyn Inc., Auburn, N. Y.
Booth 17

A complete line of diagnostic instruments for the eye, ear, nose, and throat will be demonstrated. Particularly featured will be a new headlight which is shadow free, gives brilliant illumination without reflection. It is insulated against heat and easily adjustable.

White-Haines Optical Co., Indianapolis, Ind. **Booths 21 & 22**

The Williams & Wilkins Co., Baltimore, Md. **Booth 36**

Winthrop-Stearns, Inc., New York, N. Y.
Booth 25

The members of the American Academy of Ophthalmology and Otolaryngology are cordially invited to visit the Winthrop-Stearns booth. Featured products are OpH, decongestant, astringent, antiseptic eye drops; NTZ, a combination of the time-tested, dependable decongestant Neo-Synephrine, with the potent, well-tolerated antihistaminics Thenfadil and Zephiran; Neo-Synephrine, special ophthalmic preparation for mydriasis of short duration; Pontocaine, reliable topical anesthetic with prolonged action and good penetration.

Carl Zeiss, Inc., New York, N. Y.
Booth R-16

At the forthcoming Academy meeting there will be shown for the first time the new Zeiss-Opton Ophthalmometer.

The new Zeiss-Opton Ophthalmometer incorporates for the first time with modern technical means demands made by the creator, Hermann von Helmholtz, of the first ophthalmometer which, unfortunately, have never been applied previously in ophthalmometry. The new optical system of this instrument makes possible three important features which contribute to obtain readings free from errors: (1) the mitres are projected at infinity by means of collimators for telecentric path of rays; (2) a device for determining the deviation by locating it in the plane of the focal plane of the objective, and (3) beam splitting by semi-transparent mirror squares.

Zenith Radio Corp., Chicago, Ill.
Booth 55

The Hearing Aid Division of Zenith Radio Corporation will exhibit its complete line of high-quality, low-cost hearing aids. Here is an opportunity to discover first hand what these instruments can do for the hard of hearing. Try out Zenith's **Phone Magnet**— the tiny device which brings interference-free telephone conversations by eliminating all air-borne sounds.

Zenith representatives will explain how any physician can test one of these instruments for 30 days at no cost or obligation to him or his hard of hearing patients.

Zylite Products Co., Inc., New York N. Y.
Booth 113

Zylo Ware Corp., Long Island City, N. Y.
Booth 118

SECTION ON INSTRUCTION

1 1 1

FIFTY-EIGHTH ANNUAL MEETING

American Academy of Ophthalmology and Otolaryngology

October 11-16, 1953
PALMER HOUSE
Chicago, Illinois

A. D. RUEDEMANN, M.D.
Secretary for Instruction — Ophthalmology

DEAN M. LIERLE, M.D.
Secretary for Instruction — Otolaryngology and Maxillofacial Surgery

The following condensed schedules of instruction courses are reprinted for the convenience of Fellows who have not yet placed orders for tickets.

Courses already sold out are so designated on the charts. Please do not request tickets for these courses.

For complete listing of course titles, instructors, and descriptive paragraphs about each subject, please refer to the March-April issue of the TRANSACTIONS.

General Instructions

Only members who have paid their 1953 *dues may order instruction tickets in advance of the meeting.* Members should order tickets only for their own use. Candidates for fellowship in the Academy are not privileged to reserve tickets in advance. After the session opens, no distinction between members, candidates and guests will be made in selling tickets.

Each period (1 hour) of instruction costs $2.00. *Orders unaccompanied by properly executed checks will not be filled.* Checks should be made payable to W. L. Benedict, M.D.

In case of inability to attend the meeting after order has been filled, a refund will be granted if requested prior to October 9.

If courses requested are not available and it is impossible to substitute, a refund will be granted. Refunds will not be sent by mail. A blue refund card will be placed in the Advance Registration Envelope which is received at the time of registration. This should be presented at the Instruction Desk for payment.

All orders for tickets will be filled according to postmark on envelope containing check. Telegrams will not be honored. Previous to October 1, send orders to 100 First Avenue Building, Rochester, Minnesota; after October 1, mail orders direct to W. L. Benedict, M.D., c/o Palmer House, Chicago, Illinois. Advance orders will not be filled if received after October 8.

Ordering Instruction Tickets

An *individual* course is complete in one (1) period. Most individual courses are repeated on two or three different days allowing the subscriber to choose the day on which he wishes to attend.

A *continuous* course lasts two (2) or more periods as indicated. It is not pos-

sible to purchase a ticket to only *part* of a continuous course.

At the end of this section is printed a colored sheet to be used when ordering tickets in advance. Specify preferences on this sheet and mail to the executive office. Careful attention to instructions will ensure the correct filling of your requests. *Since number of tickets for each course is limited, be sure to list second and third preferences for each period in case first preference is already sold out. In every case, effort will be made to furnish first choices.*

If you are requesting registration in the continuous courses, please specify preferences for individual courses as substitutes in order to avoid disappointment should the continuous courses which you select be sold out when your order is received.

Acknowledgment of Advance Order

As soon as an advance order is filled, a postcard, listing the courses that have been reserved for him, will be mailed to the subscriber. Please do not ask to change courses after your order has been filled. The postcard is merely an assurance that your order has been filled; it is not necessary for registration at the Academy meeting.

Claiming Instruction Tickets

No tickets will be sent through the mails. Members who have ordered instruction course tickets will register at the *Advance Order Section* of the Registration Desk during the week of the Convention to secure registration badge, tickets, and all convention material.

KEY TO DESIGNATION OF PERIODS

The periods will be designated with the following key: Select tickets for — Otolaryngology, forenoons, periods 1-2-3 — Ophthalmology, afternoons, periods 4-5-6

Day	(M)onday	(T)uesday	(W)ednesday	(Th)ursday
Period a.m.		1 2 3	1 2 3	1 2 3
Period p.m.	4 5 6	4 5 6	4 5 6	4 5 6

M-4	2:00 p.m. to 3:00 p.m.	W-4	2:00 p.m. to 3:00 p.m.
M-5	3:15 p.m. to 4:15 p.m.	W-5	3:15 p.m. to 4:15 p.m.
M-6	4:30 p.m. to 5:30 p.m.	W-6	4:30 p.m. to 5:30 p.m.
T-1	9:00 a.m. to 10:00 a.m.	Th-1	9:00 a.m. to 10:00 a.m.
T-2	10:15 a.m. to 11:15 a.m.	Th-2	10:15 a.m. to 11:15 a.m.
T-3	11:30 a.m. to 12:30 p.m.	Th-3	11:30 a.m. to 12:30 p.m.
T-4	2:00 p.m. to 3:00 p.m.	Th-4	2:00 p.m. to 3:00 p.m.
T-5	3:15 p.m. to 4:15 p.m.	Th-5	3:15 p.m. to 4:15 p.m.
T-6	4:30 p.m. to 5:30 p.m.	Th-6	4:30 p.m. to 5:30 p.m.
W-1	9:00 a.m. to 10:00 a.m.		
W-2	10:15 a.m. to 11:15 a.m.		
W-3	11:30 a.m. to 12:30 p.m.		

(Familiarity with this key will be helpful and will avoid confusion)

CHANGES IN INSTRUCTION COURSE PROGRAM AS LISTED IN THE MARCH-APRIL 1953 TRANSACTIONS

The title of Course 501, to be presented by Dr. Albert P. Seltzer on T-3 and W-3, will be "Plastic Surgery of the Nose, Abrasion of the Face, and Face-Lift Operation: New Concepts and Technics."

CONTINUOUS COURSES — OPHTHALMOLOGY

COURSE	SPEAKERS	SUBJECT	PERIODS USED
1	Allen, J. H., et al	Bacteriology & Infectious Diseases	8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5
2	Ascher	Aqueous Veins in Glaucoma	2 PERIODS M-5, M-6
3	Bedell	Medical Ophthalmoscopy	4 PERIODS M-5, T-5, W-5, Th-5
4	Berliner	.....SOLD OUT.....	
5	Birge, et al	New Theories of Vascular Disease	3 PERIODS M-4, T-4, W-4
6	Boeder	Aniseikonia & Space Eikonometry	2 PERIODS T-5, T-6
7	Brown, et al	Eye Muscles	8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5
8	Byrnes	Ophthalmic Filters	2 PERIODS M-5, M-6
9	Callahan, et al	.....SOLD OUT.....	
10	Clark	Surgery of Ocular Muscles	2 PERIODS T-5, T-6
11 or 12	Copeland	Streak Retinoscopy	4 PERIODS M-4, M-5, T-4, T-5 or W-4, W-5, Th-4, Th-5
13	Costenbader, et al	Comitant Strabismus	12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6
14	DeVoe	.....SOLD OUT.....	
15	Dunnington & Clark	.....SOLD OUT.....	
16	Fink	Management of Oblique Muscle Problems	3 PERIODS M-5, T-5, W-5
17	Friedenwald	Studies in Aqueous Flow	2 PERIODS M-4, M-5
18	Guibor	Orthoptics: Office & Home Technics	3 PERIODS T-6, W-6, Th-6
19	Hardy, et al	Basic Principles of Orthoptics	4 PERIODS M-4, T-4, W-4, Th-4
20	Henderson	Practical Perimetry	4 PERIODS M-4, M-5, T-4, T-5
21	Hughes, et al	Plastic Surgery: Advanced	8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5
22	Jardon	Fitting of Ocular Prostheses	2 PERIODS T-5, T-6
23 or 24	Johnson, L. V. & Ballintine	Aqueous Tonography	2 PERIODS M-4, M-5 or T-4, T-5
25 or 26	Johnson, V. C., & Dunlap	Diagnostic Value of X-ray	2 PERIODS M-5, M-6 or T-5, T-6

CONTINUOUS COURSES — OPHTHALMOLOGY

COURSE	SPEAKERS	SUBJECT	PERIODS USED
27	Knighton, et al	Highlights of Glaucoma	8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5
28	Kuhn	Industrial Ophthalmology	3 PERIODS M-4, T-4, W-4
29	Lloyd	Macular Lesions	2 PERIODS M-5, T-5
30	Lowrey, et al	Military Ophthalmology	2 PERIODS M-4, M-5
31	McLean, et al	.....SOLD OUT.....	
32	Meek	Surgical Anatomy of Orbit	2 PERIODS Th-4, Th-5
33	Meyer, et al	Glaucoma	8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5
34	Ogle	Essential Aspects of Binocular Vision	2 PERIODS M-5, M-6
35	O'Rourke	Manifest & Cycloplegic Refraction	2 PERIODS T-5, T-6
36	Osher	Squint Surgery	2 PERIODS W-5, W-6
37	Paton	Corneal Surgery	3 PERIODS M-6, T-6, W-6
38	Payne, et al	Practical Eye Pathology	8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5
39	Pino	Bifocal Lenses & Presbyopia	2 PERIODS M-4, T-4
40	Romaine, et al	Ocular Motility	12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6
41	Rucker	Neuro-ophthalmology	4 PERIODS M-4, T-4, W-4, Th-4
42 or 43	Schepens	Ophthalmoscopy in Diagnosis of Retinal Detachment	2 PERIODS M-5, M-6 or T-5, T-6
44	Sloane, et al	Refraction	6 PERIODS T-4, T-5, W-4, W-5, Th-4, Th-5
45	Theobald, et al	Histopathology	12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6
46	Thorpe, et al	Slit Lamp Biomicroscopy	12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6
47	Thorpe	Surgery of Intraocular Foreign Bodies	2 PERIODS T-4, W-4
48	Wheeler & Knapp	Concomitant Strabismus	6 PERIODS M-5, M-6, T-5, T-6, W-5, W-6
49	Wiener	Practical Points in Surgery	3 PERIODS M-4, T-4, W-4

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Monday

Key M-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
201	Aldrich	Psychosomatic Medicine
202	Alvaro	Uveitis: Etiology & Treatment
206	Baker	Neuralgias of Head
210	Berke	..... SOLD OUT .....
217	Chace	Ocular Malingering
218	Chamberlain	Vertical Motor Anomalies
222	Cusick	Ptosis
224	DeVoe	Corneal Dystrophies
226	Dunnington	End Results in Eye Muscle Surgery
227	Egan	Cross Cylinder Application & Theory
245	Harley	Lesions of the 3rd, 4th & 6th Cranial Nerves
250	Joy	Angiography in Ophthalmology
271	McGraw	Contact Lenses
294	Spaeth	Plastic Surgery
A	Home Study	Embryology, Anatomy & Neuro-Anatomy

Key M-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
225	Dunlap	Divergence Anomalies
230	Fralick	Surgical Anatomy of Operations for Glaucoma
233	Fry	Lesions at Posterior Pole of Eye
238	Gipner	..... SOLD OUT .....
242	Gray	Intraocular Foreign Bodies
246	Harris	Anesthetic Agents & Technics
259	Lepard	Eye Diseases Common to Children
262	Linksz	Visual Physiology
272	McGuire	Heterophoria and Heterotropia
276	Newell	Development of Refinements in Ophthalmic Surgery
296	Stimson	Lenses for the Aphakic
305	Walsh	Interesting Neuro-ophthalmologic Cases
B	Home Study	Physiology

Key M-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
205	Atkinson	Measures in Cataract Surgery
209	Berens	..... SOLD OUT .....
212	Boyes	Eye Surgery Problems
213	Burch	Psychosomatic Medicine
215	Calhoun	Dystrophies & Degenerations of Cornea
220	Chandler	..... SOLD OUT .....
223	Cutler	Procedures Used in Enucleation
228	Falls	Ocular Geriatrics
234	Gailey	Complications of Cataract Surgery
235	Gemeroy	External Diseases of Eye
236	Gibson	Retinopathies: Differential Diagnosis
240	Goar	Complications of Cataract Surgery
243	Haik	Beta Irradiation
247	Hogan	Pathology of the Choroid
248	Hughes	Plastic Surgery: Minor Procedures
256	Leinfelder	Papilledema & Optic Neuritis
261	Lewis	External Diseases
263	Lueck	Practical Optics
266	Mangold	Proper Fitting of Glasses
270	McCaslin	Ocular Anesthesia
274	Morrison	Office Management of Glaucoma
288	Rychener	Lacrimal Apparatus
C	Home Study	Optics & Principles of Biomicroscopy

SECTION ON INSTRUCTION

ADVANCE ORDER SHEET

All courses cost $2.00 for each period (one hour). Follow instructions explicitly and check for errors in listing before mailing.

Previous to OCTOBER 1

Send order to . . .

WILLIAM L. BENEDICT, M.D., *Executive Secretary-Treasurer*
American Academy of Ophthalmology and Otolaryngology
100 First Avenue Building, Rochester, Minnesota

After OCTOBER 1, mail to . . .

WILLIAM L. BENEDICT, M.D., *Executive Secretary-Treasurer*
American Academy of Ophthalmology and Otolaryngology
c/o Palmer House, Chicago, Illinois

Academy Candidates and Guests May Purchase Tickets at Registration Desk Only

(Over)

ADVANCE ORDER SHEET

ORDERS FILLED ONLY IF CURRENT DUES ARE PAID

NO REFUNDS WILL BE MADE AFTER OCTOBER 9

(Type or Print) Last Name First Name Middle Initial

Office Address

City Zone State

Days to be Present

- ☐ Monday
- ☐ Tuesday
- ☐ Wednesday
- ☐ Thursday

Total Amount Enclosed $_____

SELECTION OF COURSES ON REVERSE SIDE OF THIS BLANK

RECAPITULATION OF GENERAL INSTRUCTIONS

1. Monday, Tuesday, Wednesday, and Thursday are represented by the letters M, T, W, Th. The first period in the morning is represented by figure 1, second period by figure 2 and third period by figure 3. The first period in the afternoon is represented by figure 4, second period by figure 5, and third period by figure 6. All courses in oto-laryngology and maxillofacial surgery are presented in the mornings. All courses in ophthalmology are presented in the afternoons.
2. Reservation of instruction tickets in advance of the meeting will be made ONLY for MEMBERS whose 1953 dues have been paid. Academy candidates and guests may make selection of courses ONLY at the General Registration Desk in Chicago.
3. Make check payable to W. L. Benedict, M.D.
4. Mail order promptly to ensure reservation of first choices. If necessary, an attempt will be made to rearrange schedule to include "first choices."

(Over)

SELECTION OF COURSES

PERIOD	FIRST CHOICE		SECOND CHOICE		THIRD CHOICE	
	Course No.	Lecturer	Course No.	Lecturer	Course No.	Lecturer
MON. 4						
P.M. 5						
6						
TUES. 1						
A.M. 2						
3						
P.M. 4						
5						
6						
WED. 1						
A.M. 2						
3						
P.M. 4						
5						
6						
THURS. 1						
A.M. 2						
3						
P.M. 4						
5						
6						

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Tuesday

Key T-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
201	Aldrich	Psychosomatic Medicine
202	Alvaro	Uveitis: Etiology & Treatment
206	Baker	Neuralgias of Head
210	Berke	..... SOLD OUT .....
214	Byrnes	Adjuncts in Teaching Ophthalmology
217	Chace	Ocular Malingering
218	Chamberlain	Vertical Motor Anomalies
222	Cusick	Ptosis
224	DeVoe	Corneal Dystrophies
227	Egan	Cross Cylinder Application & Theory
229	Falls	Practical Application of Heredity
250	Joy	Angiography in Ophthalmology
271	McGraw	Contact Lenses
278	Owens & Owens	Retrolental Fibroplasia
D	Home Study	Refraction

Key T-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
225	Dunlap	Divergence Anomalies
233	Fry	Lesions at Posterior Pole of Eye
238	Gipner	Recent Advances
242	Gray	Intraocular Foreign Bodies
246	Harris	Anesthetic Agents & Technics
259	Lepard	Eye Diseases Common to Children
262	Linksz	Visual Physiology
272	McGuire	Heterophoria & Heterotropia
279	Paulson	Lighting for Comfortable & Accurate Seeing
284	Reese	Tumors & Tumor-like Lesions of the Orbit
296	Stimson	Lenses for the Aphakic
305	Walsh	Interesting Neuro-ophthalmologic Cases
E	Home Study	External Diseases, Bacteriology & Virology

Key T-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
205	Atkinson	Measures in Cataract Surgery
209	Berens	..... SOLD OUT .....
213	Burch	Psychosomatic Medicine
215	Calhoun	Dystrophies & Degenerations of Cornea
220	Chandler	..... SOLD OUT .....
223	Cutler	Procedures Used in Enucleation
234	Gailey	Complications of Cataract Surgery
235	Gemeroy	External Diseases of Eye
236	Gibson	Retinopathies: Differential Diagnosis
240	Goar	Complications of Cataract Surgery
243	Haik	Beta Irradiation
261	Lewis	External Diseases
266	Mangold	Proper Fitting of Glasses
270	McCaslin	Ocular Anesthesia
274	Morrison	Office Management of Glaucoma
283	Randolph	Handling Patients with Advanced Glaucoma
288	Rychener	Lacrimal Apparatus
289	Scheie	Emergency Eye Surgery
291	Simpson	Convergence Insufficiency & Divergence Excess
295	Steffensen	ACTH, Cortisone & Hydrocortisone
F	Home Study	Perimetry & Neuro-ophthalmology

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Wednesday

Key W-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
203	Alvis	End Results of Cataract Wound Closure
204	Amenabar	Detachment of Retina
211	Bettman	Value of Goniotomies
216	Cassady	Dacryocystitis of Infancy
237	Gifford	Dacryocystorhinostomy
241	Gordon	Surgical Importance of Orbital Fascia
254	Krewson	Paralytic Strabismus
264	Lyle	Cranial Lesions
265	Macnie	Head Trauma with Ocular Significance
269	Martens	Problems in Presbyopia
273	Meyer	Glaucoma Surgery
281	Pfeiffer	Diagnosis with Aid of X-Ray
282	Post	Operating Room Technic
298	Tabor	Vertical Muscles
301	Theodore	Ocular Therapeutics
G	Home Study	Extraocular Muscles

Key W-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
219	Chamlin	Perimeter: Types & Technics
232	Fread	Ideal Refraction Equipment
239	Givner	Systemic Disease
244	Hanser	Studies in Ocular Allergy
249	Jobe	Refraction
251	Katz	Corneal Dystrophies
257	Lemoine	Vitreous Opacities
275	Mullen	Ophthalmic Emergencies
277	Olson	Iritis, Iridocyclitis & Keratitis
280	Perera	Retinal Detachment
286	Rudolph	Advantages of General Anesthesia
292	Smith	Practical Points in Office Procedure
293	Spaeth	Lacrimal Gland & Sac
300	Tassman	Retinopathy in Diabetics
303	Trueman	Cerebral Lesions
304	Veirs	..... SOLD OUT .....
306	Wendland	Chiasmal Lesions
H	Home Study	Ophthalmoscopy & Medical Ophthalmology

Key W-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
208	Bellows	Cataract
212	Boyes	Eye Surgery Problems
221	Cholden	Informing Patient of Blindness: Psychiatric Aspects
252	Klien	Clinical Pathology of Retinal Lesions
255	Kronfeld	..... SOLD OUT .....
256	Leinfelder	Papilledema & Optic Neuritis
258	Leopold	..... SOLD OUT .....
260	Levy	Motion Picture Photography: Methods & Highlights
267	Mann, M.	Hypertensive Vascular Changes in Fundus
268	Mann, W.	Optical Correction of Aphakia
285	Robinson	Reading Retardation & Vision
287	Rundles	Streak Retinoscopy
290	Scheie	Surgery in Congenital Glaucoma
295	Steffensen	ACTH, Cortisone & Hydrocortisone
297	Swan	Ophthalmic Therapeutics
299	Tait	Accommodation & Convergence Abnormalities
307	Whalman	Minor Surgical Procedures of Eye
308	Wood	Macular Lesions
I	Home Study	Pathology

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Thursday

Key Th-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
203	Alvis	End Results of Cataract Wound Closure
204	Amenabar	Detachment of Retina
211	Bettman	Value of Goniotomies
216	Cassady	Dacryocystitis of Infancy
237	Gifford	Dacryocystorhinostomy
241	Gordon	Surgical Importance of Orbital Fascia
253	Kornzweig	Practical Embryology
254	Krewson	Paralytic Strabismus
264	Lyle	Cranial Lesions
265	Macnie	Head Trauma with Ocular Significance
269	Martens	Problems in Presbyopia
281	Pfeiffer	Diagnosis with Aid of X-Ray
282	Post	Operating Room Technic
298	Tabor	Vertical Muscles
301	Theodore	Ocular Therapeutics
J	Home Study	Pharmacology & Therapeutics

Key Th-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
207	Barkan	..... SOLD OUT .....
208	Bellows	Cataract
232	Fread	Ideal Refraction Equipment
239	Givner	Systemic Disease
244	Hanser	Studies in Ocular Allergy
249	Jobe	Refraction
251	Katz	Corneal Dystrophies
257	Lemoine	Vitreous Opacities
275	Mullen	Ophthalmic Emergencies
277	Olson	Iritis, Iridocyclitis & Keratitis
280	Perera	Retinal Detachment
286	Rudolph	Advantages of General Anesthesia
292	Smith	Practical Points in Office Procedure
300	Tassman	Retinopathy in Diabetics
303	Trueman	Cerebral Lesions
304	Veirs	..... SOLD OUT .....
306	Wendland	Chiasmal Lesions

Key Th-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
252	Klien	Clinical Pathology of Retinal Lesions
255	Kronfeld	..... SOLD OUT .....
267	Mann, M.	Hypertensive Vascular Changes in Fundus
268	Mann, W.	Optical Correction of Aphakia
285	Robinson	Reading Retardation & Vision
287	Rundles	Streak Retinoscopy
290	Scheie	Surgery in Congenital Glaucoma
295	Steffensen	..... SOLD OUT .....
299	Tait	Accommodation & Convergence Abnormalities
307	Whalman	..... SOLD OUT .....
308	Wood	Macular Lesions

CONTINUOUS COURSES AND PANEL DISCUSSIONS
OTOLARYNGOLOGY

COURSE	SPEAKERS	SUBJECT	PERIODS USED
101	Altmann & Waltner	Histopathology of Ear—Part I	2 PERIODS W-1, W-2
102	Altmann & Waltner	Histopathology of Ear—Part II	2 PERIODS Th-1, Th-2
103 or 104	Boies, et al	Head Pain	2 PERIODS W-2, W-3 or Th-2, Th-3
105 or 106	Bryan & Bryan	Cytologic Pathology in the Differential Diagnosis of ENT Diseases	2 PERIODS T-1, T-2 or W-2, W-3
107 or 108	Clerf, et al	Diseases of the Larynx, Neck, Tracheobronchial Tree & Esophagus	2 PERIODS W-1, W-2 or Th-1, Th-2
109 or 110	Conley, et al	Cancer of Head & Neck	2 PERIODS T-1, T-2 or Th-1, Th-2
111	Converse		..... SOLD OUT .....
112	Converse		..... SOLD OUT .....
113	Cullen, et al	Anesthesia for Otolaryngologic Surgery	2 PERIODS Th-1, Th-2
114	Fowler & Montreuil	Histology of Ear — Vertical Sections	2 PERIODS T-1, T-2
115	Fowler & Montreuil	Histology of Ear — Horizontal Sections	2 PERIODS W-1, W-2
116	Furstenberg, et al	Tumors & Infections of the Neck	2 PERIODS T-2, T-3
117 or 118	Hansel	Allergy	2 PERIODS W-2, W-3 or Th-2, Th-3
119	Hoople, et al	Fluid Ears	2 PERIODS Th-2, Th-3
120	Jackson	Peroral Endoscopy	3 PERIODS T-1, W-3, Th-1
121	Kazanjian, et al	Traumatic Injuries of Face	2 PERIODS Th-1, Th-2
122 or 123	Lindsay	Vertigo	2 PERIODS T-1, T-2 or W-1, W-2
124 or 125	Marcus, et al	Hearing Problems in Children	2 PERIODS T-1, T-2 or Th-1, Th-2
126 or 126-A	Meltzer, et al	Otological Problems	2 PERIODS T-1, T-2 or W-1, W-2
127 or 128	Moses	Speech & Voice Therapy	2 PERIODS T-1, T-2 or W-1, W-2
129	Mueller, et al	Hearing Aids	2 PERIODS W-1, W-2
130	Priest, et al	Medication for the Otolaryngologist	1 PERIOD T-1
131	Proetz, et al	Rhinological Problems	2 PERIODS W-2, W-3
132 or 133	Scheldrup	Anatomy of the Neck	2 PERIODS T-2, T-3 or W-2, W-3
134	Semenov	Therapy in Pathology of ENT	2 PERIODS Th-1, Th-2
135	Senturia, et al	External Otitis	2 PERIODS T-1, T-2
136	Tucker, et al	Esophageal Diseases & Foreign Bodies	2 PERIODS T-1, T-2
137	Tucker, et al	Laryngeal Surgery	2 PERIODS W-1, W-2

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Tuesday

Key T-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
402	Barone	Hay Fever
404	Bellucci	Submucous Resection of Nasal Septum
411	Carpenter & Goff	Respiratory and Swallowing Problems in the Newborn
412	Cody	The Elusive Facial Nerve
420	Day	What the Otologist Can Do for Deafness
423	Engler & Hulse	Tuberculosis of the Larynx
442	Holinger	Pediatric Laryngology
445	Hosler	Cardiac Arrest
449	Huffman	Zygomatic Fracture-Dislocations
453	Jones, L. T.	Tear Sac Surgery
461	Kinney	Acoustic Trauma
465	Lederer	Diagnostic Approaches & Recent Trends
468	Lempert	Temporal Bone Surgery
475	McQuiston	Endaural Radical Mastoidectomy
484	Pastore	A Lump in the Throat
498	Schall	Cancer of the Nose & Sinuses
499	Schuknecht	Carcinoma of the Oral Cavity & Jaws
504	Simmons	Intranasal Dacryocystorhinostomy
505	Simonton	Recruitment of Loudness and Diplacusis Binauralis
509	Sullivan	Facial Paralysis and Bell's Palsy
511	Tingwald	Tumors of the Paranasal Sinuses
514	Van Alyea	Chronic Sinusitis

Key T-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
406	Boyden	Chronic Frontal Sinus Surgery
415	Corgill	Laryngopharyngectomy & Bilateral Dissection
428	Glick	The Sinuses
432	Hallberg	..... SOLD OUT .....
435	Harris	Poliomyelitis: Respiratory Complications
439	Hilger	Therapeutic Approaches to the Inner Ear
447	House	Atresia of the Ear Canal
450	Ireland	Common Sense Treatment of Office Patients
454	Jordan, R. E.	Allergy in Otology
460	Kemler	Mirror Laryngoscopy
466	LeJeune	Intralaryngeal Surgery
472	McGannon	Respiratory Allergy
476	Miller	Laryngeal Emergencies in Infants & Children
480	Nash	Otolaryngologic Mishaps
485	Pearlman	ENT Complaints in Old Age
488	Putney	Surgery of Neck Tumors
490	Reger	Detection of Auditory Malingering
496	Rosenberger	Practical Aspects of Childhood Otology
500	Sellers	Headache
515	Walsh	Acute & Chronic Otitis Media
517	Work	Wide Field Laryngectomy

Key T-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
401	Ashley	Dust & Other Perennial Inhalant Therapy
405	Bordley	Hearing Disorders in Preschool Children
407	Brandenburg	..... CANCELLED .....
410	Carhart	Audiogram in Otologic Diagnosis
414	Cohen	Rhinoplasty Complications
419	Davison	Bronchology & Bacterial Allergy
421	Derlacki	Role of Allergy in Deafness
427	Forman	..... CANCELLED .....
430	Glorig	Construction of a Practical Soundproof Room
433	Hampsey	Respiratory Allergy
434	Harrill	Bronchography
438	Hilger	Direct Fixation of Facial Fractures
440	Hill	Antro-Alveolar Fistulas
443	Holinger	Esophageal Diseases in Infants & Children
444	Holmes	Scars of the Face
451	Johnson	Anesthesia in Adult Tonsillectomies
456	Juers	Hearing Tests
463	Lake	Chronic Suppurative Otitis Media
467	Lempert	Diagnosing Type of Hearing Loss
470	Macgregor	Psychological Hazards of Plastic Surgery of the Face
471	Maxwell	Tumors of the Face & Neck in Infancy & Childhood
477	Missal	Skin Tests in Allergy
478	Moore	Eustachian Tube Problems
486	Perlman	Serous Otitis
487	Proetz	Clinical Aspects of Nasal Respiration
492	Richards	Suggestions Concerning Office Practice
501	Seltzer	Plastic Surgery
502	Shambaugh	Practical Office Routine in Otologic Problems
510	Thornell	Intralaryngeal Arytenoidectomy
516	Weille	Nasal & Sinus Surgery in Respiratory Allergy

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Wednesday

Key W-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
402	Barone	Hay Fever
403	Becker	Plastic Surgery of Protruding Ears
404	Bellucci	..... SOLD OUT .....
411	Carpenter & Goff	Respiratory and Swallowing Problems in the Newborn
412	Cody	The Elusive Facial Nerve
416	Costen	Mandibular Joint Syndrome
418	Crispell	Allergy in Children
419	Davison	Bronchology & Bacterial Allergy
420	Day	What the Otologist Can Do for Deafness
423	Engler & Hulse	Tuberculosis of the Larynx
431	Goodhill	..... SOLD OUT .....
437	Hickey	Functional or Organic Nervous Disease
440	Hill	Antro-Alveolar Fistulas
442	Holinger	Pediatric Laryngology
449	Huffman	Zygomatic Fracture-Dislocations
453	Jones, L. T.	Tear Sac Surgery
454	Jordan, R. E.	Allergy in Otology
474	McNally	Labyrinthine Tests
479	Morrison	Laryngeal Paralysis
489	Rawlins	ACTH, Cortisone & the Antihistaminic Drugs
495	Robison	Otitis Media with Effusion
498	Schall	Cancer of the Nose & Sinuses
499	Schuknecht	Carcinoma of the Oral Cavity & Jaws
503	Shuster	Vestibular Apparatus: Clinical Considerations
508	Slaughter	Blood Vessel Tumors of Head & Neck
512	Tremble	Practical Therapeutics in Office Practice

Key W-2 — 10:15 a.m. to 11:15 a.m.

Course No	SPEAKER	SUBJECT
406	Boyden	Chronic Frontal Sinus Surgery
415	Corgill	Laryngopharyngectomy & Bilateral Dissection
425	Fabricant	Modern Medication of Nose & Sinuses
426	Figi	Tumors of Ear & Mastoid
432	Hallberg	Severe Nosebleed
436	Hedgecock	Advanced Clinical Audiometry
448	Howard	Office Procedures in ENT
450	Ireland	Common Sense Treatment of Office Patients
455	Jordan, V.M.	Sudden Death in the Operating Room
458	Kelemen	Tonsillar Tissue
462	Kos	Clinical Orientation in Management of Dizziness
467	Lempert	Diagnosing Type of Hearing Loss
482	Neivert	Workshop for Deformed Instruments
493	Rinkel	Food Allergy in Otolaryngologic Practice
497	Ryan	Headache
500	Sellers	Headache
505	Simonton	Recruitment of Loudness & Diplacusis Binauralis
506	Singleton	Acute Otitis Media
515	Walsh	Acute & Chronic Otitis Media
518	Wynn	Facial Injuries: Plastic Repair

Key W-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
401	Ashley	Dust and Other Perennial Inhalant Therapy
405	Bordley	Hearing Disorders in Preschool Children
407	Brandenburg	..... CANCELLED .....
408	Brodnitz	The Actor & Singer in ENT Practice
409	Canfield	Audiology for Practicing Otologist
417	Craft	Dermatologic Allergy
422	DeWeese	The Dizzy Patient
424	Erich	Fractures of Facial Bones
427	Forman	..... CANCELLED .....
433	Hampsey	Respiratory Allergy
441	Hilsinger	Vascular Headaches
444	Holmes	Scars of the Face
446	House	Surgery of Chronically Discharging Ear
452	Jones, A. C.	Helpful Hints for Everyday Practice
457	Keaster	Speech & Hearing in Children Under Five
463	Lake	Chronic Suppurative Otitis Media
464	Lassman	Prescription of Hearing Aids
469	Levin	Management of Defects of Speech & Voice
470	Macgregor	Psychological Hazards of Plastic Surgery of the Face
473	McLaurin	Otitis Externa: Therapy
483	Ogura	Laryngectomy & Neck Dissection
492	Richards	Suggestions Concerning Office Practice
494	Roberts	Medical Otolaryngology
501	Seltzer	Plastic Surgery
509	Sullivan	Facial Paralysis and Bell's Palsy
510	Thornell	Intralaryngeal Arytenoidectomy

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Thursday

Key Th-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
403	Becker	Plastic Surgery of Protruding Ears
413	Cohen	Deviated Noses & Their Correction
416	Costen	Mandibular Joint Syndrome
418	Crispell	Allergy in Children
428	Glick	The Sinuses
431	Goodhill	Management of Tinnitus
437	Hickey	Functional or Organic Nervous Disease
439	Hilger	Therapeutic Approaches to the Inner Ear
443	Holinger	Esophageal Diseases in Infants & Children
452	Jones, A. C.	Helpful Hints for Everyday Practice
460	Kemler	Mirror Laryngoscopy
466	LeJeune	Intralaryngeal Surgery
471	Maxwell	Tumors of the Face & Neck in Infancy & Childhood
472	McGannon	Respiratory Allergy
474	McNally	Labyrinthine Tests
475	McQuiston	Endaural Radical Mastoidectomy
478	Moore	Eustachian Tube Problems
483	Ogura	Laryngectomy & Neck Dissection
484	Pastore	A Lump in the Throat
489	Rawlins	SOLD OUT
493	Rinkel	Food Allergy in Otolaryngologic Practice
503	Shuster	Vestibular Apparatus: Clinical Considerations
504	Simmons	Intranasal Dacryocystorhinostomy
507	Skolnik	Cleft Palate
508	Slaughter	Blood Vessel Tumors of Head & Neck
513	Van Alyea	Embryology of ENT

Key Th-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
425	Fabricant	Modern Medication of Nose & Sinuses
426	Figi	Tumors of Ear & Mastoid
435	Harris	Poliomyelitis: Respiratory Complications
445	Hosler	Cardiac Arrest
447	House	Atresia of the Ear Canal
455	Jordan, V.M.	Sudden Death in the Operating Room
459	Kelemen	Mechanical Injuries of Hearing Organ
462	Kos	Clinical Orientation in Management of Dizziness
465	Lederer	Diagnostic Approaches & Recent Trends
476	Miller	Laryngeal Emergencies in Infants & Children
482	Neivert	Workshop for Deformed Instruments
485	Pearlman	ENT Complaints in Old Age
487	Proetz	Clinical Aspects of Nasal Respiration
491	Reger	Practical Office Audiometry
495	Robison	Otitis Media with Effusion
496	Rosenberger	Practical Aspects of Childhood Otology
502	Shambaugh	Practical Office Routine in Otologic Problems
506	Singleton	Acute Otitis Media
511	Tingwald	Tumors of the Paranasal Sinuses
512	Tremble	Practical Therapeutics in Office Practice
517	Work	Wide Field Laryngectomy

Key Th-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
408	Brodnitz	The Actor & Singer in ENT Practice
409	Canfield	Audiology for Practicing Otologist
410	Carhart	Audiogram in Otologic Diagnosis
417	Craft	Dermatologic Allergy
421	Derlacki	Role of Allergy in Deafness
422	DeWeese	The Dizzy Patient
424	Erich	Fractures of Facial Bones
429	Glorig	Terms Used in Audiology
434	Harrill	Bronchography
436	Hedgecock	Advanced Clinical Audiometry
441	Hilsinger	Vascular Headaches
446	House	Surgery of Chronically Discharging Ear
448	Howard	Office Procedures in ENT
451	Johnson	Anesthesia in Adult Tonsillectomies
456	Juers	Hearing Tests
461	Kinney	Acoustic Trauma
464	Lassman	Prescription of Hearing Aids
468	Lempert	Temporal Bone Surgery
473	McLaurin	Otitis Externa: Therapy
477	Missal	Skin Tests in Allergy
479	Morrison	Laryngeal Paralysis
481	Nash	Functional Diseases of Nose
486	Perlman	Serous Otitis
488	Putney	Surgery of Neck Tumors
494	Roberts	Medical Otolaryngology
497	Ryan	Headache
518	Wynn	Facial Injuries: Plastic Repair

News - - - Notes

XVII INTERNATIONAL CONGRESS OF OPHTHALMOLOGY

The International Council of Ophthalmology has decided to extend the scope of the XVII International Congress of Ophthalmology by a two-day session in Montreal, Canada. This will take place on September 10 and 11, 1954. Those who attend both meetings will travel to New York on September 12 and the Opening Ceremony of that part of the Congress will be held in the Grand Ballroom of the Waldorf-Astoria Hotel on Monday morning, September 13. The New York session will continue through Friday, September 17. Details of the two programs will be announced later. A single registration fee will cover both the Montreal and the New York sessions of the Congress. Administrative affairs of the Congress will continue to be handled through the office of the Secretary General, Dr. William L. Benedict, 100 First Avenue Building, Rochester, Minnesota, U.S.A.

AMERICAN BOARD OF OPHTHALMOLOGY

The American Board of Ophthalmology will conduct examinations October 5 to 9, 1953, at the Palmer House, Chicago, Ill.

AMERICAN BOARD OF OTOLARYNGOLOGY

The American Board of Otolaryngology will conduct examinations October 5 to 9, 1953, at the Palmer House, Chicago, Ill.

AMERICAN OPHTHALMOLOGICAL SOCIETY

At the annual meeting of the American Ophthalmological Society the following officers were elected: president, Dr. William L. Benedict; vice-president, Dr. Everett L. Goar; secretary-treasurer, Dr. Maynard C. Wheeler; and editor, Dr. Gordon M. Bruce.

The next annual meeting of the American Ophthalmological Society will be held on June 16, 17 and 18, 1954 at the Many Glacier Hotel, Glacier Park, Mont.

THE AMERICAN ORTHOPTIC COUNCIL

The American Orthoptic Council will hold its annual meeting at 7:00 p.m. on Saturday, October 10, 1953, at the Palmer House, Chicago.

INTERNATIONAL ASSOCIATION OF EYE, EAR, NOSE AND THROAT SOCIETY SECRETARIES

The International Association of Eye, Ear, Nose and Throat Society Secretaries will hold its annual meeting at 5:30 p.m. on Tuesday evening, October 13, 1953, in Private Dining Room 18 of the Palmer House, Chicago. The American Academy of Ophthalmology and Otolaryngology will be host to the Secretaries at dinner following the meeting.

THE SOCIETY OF MILITARY OPHTHALMOLOGISTS

The Society of Military Ophthalmologists is planning a cocktail party and dinner on Tuesday evening, October 13, at the Palmer House, Chicago, during the Academy meeting.

GEORGIA

The spring meeting of the Georgia Society of Ophthalmology and Otolaryngology will be held March 5 and 6, 1954, at the General Oglethorpe Hotel, Savannah, Ga. The speakers will be: Dr. Paul Chandler, Boston; Dr. A. B. Reese, New York; Dr. Henry P. Wagener, Rochester, Minn.; Dr. L. R. Boies, Minneapolis; Dr. Francis LeJeune, New Orleans; and Dr. J. H. Maxwell, Ann Arbor.

LOUISIANA

The winter convention of The New Orleans Academy of Ophthalmology will be held during the first week of February 1954. The subject of the symposium will be Bacteriology and Infectious Diseases. Further information may be secured through Dr. James Allen or Dr. George Haik, New Orleans, La.

VIRGINIA

The fall clinical session of the Virginia Society of Ophthalmology and Otolaryn-

gology will be held in Richmond, Va., on November 30 to December 3, 1953. The spring meeting will be held at the Chamberlain Hotel in Old Point Comfort, Va., May 7 and 8, 1954.

CANADA

The Faculty of Medicine of the University of Toronto offers a Refresher Course in Eye Surgery from April 20 to 26, 1954. The instruction will consist of lectures, operative clinics on patients and cadaver surgery in small groups. The guest surgeons will be John Foster, M.B., F.R.C.S., Leeds, England, and Wendell L. Hughes, M.D., F.A.C.S., Hempstead, New York. The staff of the Department of Ophthalmology in the University will contribute extensively to the course.

The course will be given for a minimum of 10 students and a maximum of 30 students. Applications should be made to the Dean of the Faculty of Medicine not later than January 31, 1954.

The Sixteenth Annual Meeting of the Canadian Ophthalmological Society was held at Minaki Lodge, Minaki, Ontario, in June 1953. Dr. Kenneth B. Johnston was in the chair.

This being Coronation year the Society, among other national societies, was invited to nominate one of its members to receive the Coronation Medal. The President, Dr. Kenneth B. Johnston of Montreal, was elected to receive this honor.

The Society went on record as being in favor of reciprocity with the American Academy of Ophthalmology and Otolaryngology. In fact it was pointed out that in a revision of the Constitutional in 1951, the American Board was placed in the same category as Certification by the Royal College of Physicians and Surgeons of Canada, as an entrance requirement to the Society.

The Scientific Program consisted of 16 papers and case reports covering a wide field of ophthalmology. Prominent among these were glaucoma, retrolental fibroplasia and macular edema following cataract extraction.

Dr. Edward P. Burch of St. Paul, Minnesota, was the guest of honor. He presented a paper on "Safeguards in Cataract Surgery" which covered the subject exhaustively and was very much appreciated.

Dr. Mark Robert Marshall of Edmonton, Alberta, was elected President for the coming year, while Dr. P. B. Macfarlane of Hamilton, Ontario, was elected Vice-President. The Secretary, Dr. R. G. C. Kelly, Toronto; the Treasurer, Dr. J. V. V. Nicholls, Montreal; and the Editor-in-Chief of the Transactions, Dr. Clement McCulloch of Toronto, were re-elected.

GERMANY

The Deutsche Ophthalmologische Gesellschaft Heidelberg will meet in Heidelberg from August 30 to September 2, 1953.

POSITIONS AVAILABLE

Wanted: Ophthalmologist desires practice either private, with a group, or in association with an otolaryngologist. Has completed Junior residency including basic sciences. Married. Army service completed. For further information please write to Dr. Harry M. Katz, 45 Elyton Court, Birmingham, Alabama.

Listings of positions available should be sent to W. L. Benedict, M.D., 100 First Avenue Building, Rochester, Minn., by the fifth of the month preceding publication. They should include (1) type of physician wanted (ophthalmologist or otolaryngologist; diplomate, resident, etc.); (2) type of position to be filled; and (3) whom to write for further information. Unless otherwise requested, listings will be published once only.

The Academy will handle no further correspondence beyond the listing and assume no responsibility. Neither does it endorse or guarantee any of the published listings.

AVAILABLE SUPPLIES

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	Price
RETINAL DETACHMENT, H. Arruga, et al	$1.00
OCULAR SURGERY, Conrad Berens and Michel Loutfallah	1.00
CATARACT TYPES, F. C. Cordes	1.00
OUTLINE OF NEURO-OPHTHALMOLOGY, P. J. Leinfelder	1.00
A COMPREHENSIVE DESCRIPTION OF THE ORBIT, ORBITAL CONTENT, AND ASSOCIATED STRUCTURES WITH CLINICAL APPLICATIONS, R. E. McCotter et al	1.00
GLAUCOMA, Samuel J. Meyer	1.00
THE INTERPRETATION OF VISUAL FIELDS (Revised), C. W. Rucker	1.00
DISTURBANCES OF OCULAR MOTILITY, Richard G. Scobee	1.00
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NONSURGICAL COMPLICATIONS OF OTOLARYNGOLOGIC SURGERY, J. J. Pressman et al	1.00

MONOGRAPHS

HELPFUL HINTS FOR HANDLING THE HEARING HANDICAPPED, Gordon D. Hoople et al	$0.25
HEARING TESTS (Revised), Arthur L. Juers	0.25
THE EMBRYOLOGY OF THE EAR, NOSE, AND THROAT, O. E. Van Alyea	0.25
STRABISMUS: A SYMPOSIUM, Kenneth C. Swan et al	0.50
REFRACTION DIFFICULTIES (Revised) Clarence A. Veasey, Jr.	0.50

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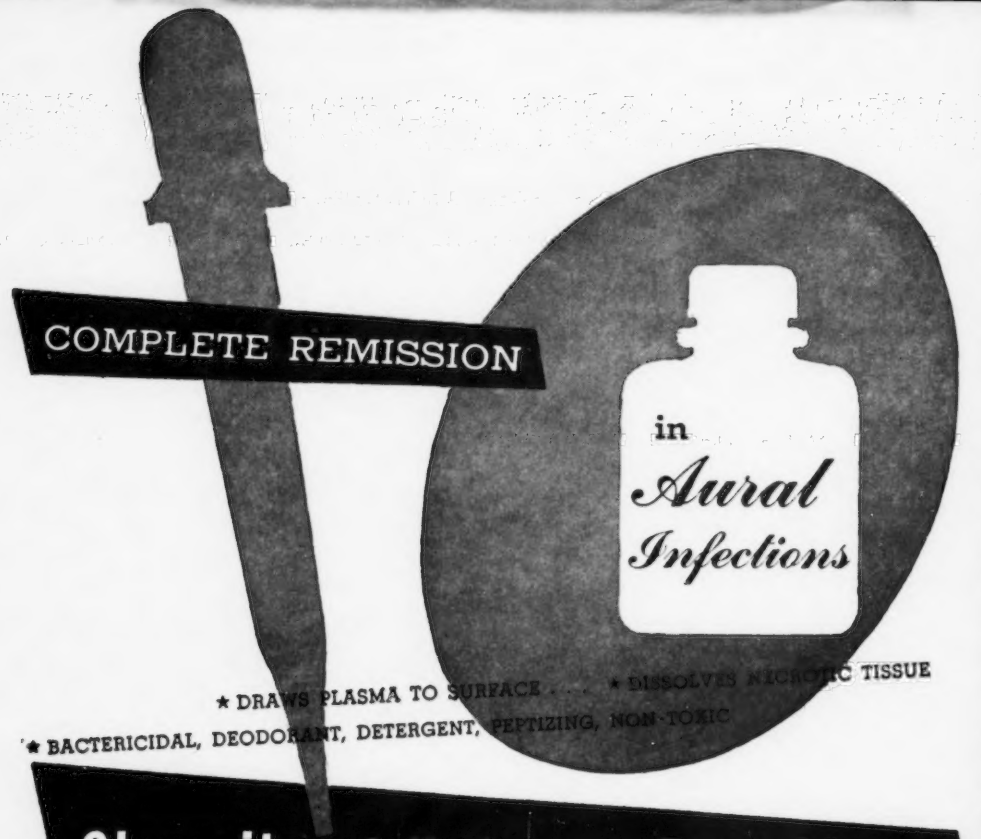

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★ BIBLIOGRAPHY

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E., E., N., & T. Mo., 26:27, 1947.
Laryngoscope, 56:556, 1946.
New Eng. J. Med., 234:468, 1946.
Annals of Allergy, 4:33, 1946.
J. A. Ph., A., (Sc. Ed.) 35:304, 1946.

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